# MEDICINE

ESSENTIALS FOR PRACTITIONERS AND STUDENTS

*ALSO BY DR BEAUMONT*

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## ESSENTIALS FOR PRACTITIONERS AND STUDENTS

By

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*SEVENTH EDITION*

WITH 70 ILLUSTRATIONS



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# PREFACE TO THE SEVENTH EDITION

In preparing this the seventh edition I have subjected the book to a thorough revision in order to include the many advances made in medicine during the past four years

New articles have been written on the following subjects Behçet s syndrome gastric diverticula the dumping syndrome carcinoid tumours of the intestine steatorrhœa proctalgia fugax thrombosis of the hepatic veins post necrotic hepatic cirrhosis constitutional hyperbilirubinæmia hepatic coma the post cholecystectomy syndrome sequestration of a lung North American blastomycosis the Wolff Parkinson White syndrome ischæmic heart disease dissecting aneurysm tuberous sclerosis cervical spondylosis Christmas disease agammaglobulinæmia sarcoidosis histoplasmosis osteoporosis kwashiokor primary amyloidosis the Fanconi syndrome idiopathic hypoparathyroidism and pseudo hypoparathyroidism Chapter VI on Water and Electrolytic Balance is new and has been kindly written for me by Dr P H Friedlander

The following sections have been largely or entirely rewritten Fibrocystic disease of the pancreas suppurative bronchitis the etiology of carcinoma of the lung the pneumonias the types of pulmonary tuberculosis the etiology of emphysema collapse of the lung anticoagulants abscess of the lung cor pulmonale the signs of mitral stenosis arteriosclerosis high blood pressure Raynaud s disease tuberculous meningitis epilepsy nephritis nephrosis uræmia and the etiology of pernicious anæmia

The treatment of the following subjects has been recast Gastric ulcer cœliac disease acute pancreatitis ascites ruptured œsophageal veins pulmonary tuberculosis emphysema meningococcal meningitis neuro syphilis the prophylaxis of measles and whooping cough the treatment of scarlet fever typhoid fever syphilis tetanus gout malaria chronic leukæmia and rheumatoid arthritis

Notes have been added on the following subjects Calcification of the liver liver palms vascular spiders silver stools cough syncope pre menstrual asthma printers asthma the rheumatoid lung hamartoma of lung diffuse interstitial pulmonary fibrosis endomyocardial fibrosis the auriculo temporal syndrome the Klippel Feil syndrome cation exchange resins pseudohæmophilia thrombasthenia cat scratch disease potassium losing nephritis Conn s syndrome the Rogitine test for phæchromocytoma and the insulin zinc suspensions

Other additions include the therapeutic use of over thirty new preparations Six figures have been omitted and seven new figures added

I have received much help in preparing this edition from my publishers Messrs J and A Churchill and in particular from Mr J Rivers My secretary Miss E G Smyth has made many valuable suggestions has prepared the manuscript for the press and has helped me with the proof sheets I am indebted to Miss Josephine Hart for the photograph of my patient suffering from insulin lipodystrophy (Fig 64) and to Mr J Shields for preparing the index of the book

London G E Beaumont

# PREFACE TO THE FIRST EDITION

It has been my endeavour to produce a text book of medicine which is not too long for the use of the student preparing for his final examination and which will also be of assistance to the general practitioner The ideal aimed at has been to include the essentials of medicine and to omit all ex traneous matter to give the student a clear account of the essential features of each disease described and to supply the practitioner with information as to the investigations required to establish the diagnosis in any particular disease together with an up to date account of a definite line of treatment

Special attention is directed to clinical findings Several detailed diet sheets are given such as those suitable for the treatment of pneumonia typhoid fever diabetes nephritis gastric ulcer obesity constipation etc The appropriate dosage prescriptions and methods of administration of drugs are included in the treatment sections of the various diseases over one hundred prescriptions being given in full No effort has been spared to bring every article up to date to illustrate them with explanatory diagrams figures and temperature charts and to connect them with cross references A series of diagrams illustrating the anatomy and physiology of the parts concerned has been introduced into the chapter dealing with nervous diseases so that this difficult branch of medicine may be more easily understood The old anatomical terminology has been employed but a glossary showing the corresponding terms in the international (B N A ) nomenclature is included at the beginning of the book

*It is still the duty of the general physician attached to the teaching staff* of a general hospital to care for patients suffering from most branches of medical diseases and to instruct students in the symptoms signs and treat ment of such diseases Such is the reply if reply be needed to the criticism *that the day of the one man text book has passed If this is so it could be* argued that no single physician should be allowed to teach general medicine and have charge of general medical wards

Psychological and Dermatological medicine have not been included They *are highly specialised subjects which are not dealt with in the general medical* wards of a hospital It is true that the student and practitioner must have a working knowledge of these branches of medicine but this is best acquired from practical experience in the special departments of a general hospital Infectious fevers have been included as they frequently cause difficulty in the diagnosis of other medical diseases and they are so important in general practice Apart from this the diseases described are those treated in the medical wards of a general hospital

This book is largely based upon personal experience in hospital and private practice and I am indebted first of all to my teachers and secondly to the authors of the numerous books and articles which I have read I should like to acknowledge them in detail but space does not permit and I take this opportunity of thanking them all for the information they have put before the medical profession

It is a great pleasure to express my gratitude to Dr Lee Lander who has read the typescript and made valuable suggestions and alterations The publishers of this volume have rendered me every facility and assistance they have been patient during the five years it has taken me to write and rewrite the manuscript and I cannot thank them sufficiently for their help and courtesy

G F BEAUMONT

LONDON 1932

## CONTENTS

# ANATOMICAL GLOSSARY

| Old Nomenclature | International (B N.A ) Nomenclature |
|---|---|
| Fissure of Rolando | Central sulcus |
| Sylvian fissure | Lateral cerebral fissure |
| Lenticular nucleus | Lentiform nucleus |
| Foramen of Monro | Interventricular foramen |
| Foramen of Majendie | Medial aperture |
| Foramen of Luschka | Lateral aperture |
| Sylvian aqueduct | Cerebral aqueduct |
| Valve of Vieussens | Anterior medullary velum |
| Crus cerebri | Cerebral peduncle |
| Superior corpus quadrigeminum | Superior colliculus |
| Inferior corpus quadrigeminum | Inferior colliculus |
| Superior cerebellar peduncle | Brachium conjunctivum |
| Middle cerebellar peduncle | Brachium pontis |
| Inferior cerebellar peduncle | Restiform body |
| Cisterna magna | Cisterna cerebello medullaris |
| Cisterna basalis | Cisterna interpeduncularis |
| Superior longitudinal sinus | Superior sagittal sinus |
| Inferior longitudinal sinus | Inferior sagittal sinus |
| Circular sinus | Intercavernous plexus |
| Basilar sinus | Basilar plexus |
| Lateral sinus | Transverse sinus |
| Pacchionian bodies | Arachnoideal granulations |
| Gasserian ganglion | Semilunar ganglion |
| Ganglion of Scarpa | Vestibular ganglion |
| Deiters nucleus | Lateral vestibular nucleus |
| Column of Goll | Funiculus gracilis |
| Column of Burdach | Funiculus cuneatus |
| Direct pyramidal tract | Anterior cerebro spinal tract |
| Crossed pyramidal tract | Lateral cerebro spinal tract |
| Direct cerebellar tract | Posterior spino cerebellar tract |
| Indirect cerebellar tract | Anterior spino-cerebellar tract |
| Clarke s column | Dorsal nucleus |
| Circumflex nerve | Axillary nerve |
| Lesser internal cutaneous nerve | Medial cutaneous nerve |
| Intercosto-humeral nerve | Intercosto brachial nerve |
| External cutaneous nerve (arm) | Posterior cutaneous nerve |
| Internal cutaneous nerve (arm) | Medial cutaneous nerve |
| Musculo spiral nerve | Radial nerve |
| Posterior interosseous nerve | Deep branch of radial nerve |
| Radial nerve | Superficial branch of radial nerve |
| External cutaneous nerve (leg) | Lateral cutaneous nerve |
| Internal cutaneous nerve (leg) | Medial cutaneous nerve |
| Middle cutaneous nerve (leg) | Intermediate cutaneous nerve |
| Small sciatic nerve | Posterior cutaneous nerve |
| Peroneal nerve | Common peroneal nerve |
| Anterior crural nerve | Femoral nerve |
| Genito-crural nerve | Genito-femoral nerve |
| Internal saphenous nerve | Saphenous nerve |
| Internal popliteal nerve | Tibial nerve |
| Anterior tibial nerve | Deep peroneal nerve |
| External saphenous nerve | Sural nerve |
| Musculo cutaneous nerve | Superficial peroneal nerve |
| Internal plantar nerve | Medial plantar nerve |
| External plantar nerve | Lateral plantar nerve |

# BLOOD

## Normal Chemical Constituents

FIGURES are mg /100 ml unless indicated otherwise mEq /L = milli equivalent per litre This is one thousandth of a gramme equivalent which is the amount of a compound which can react with or be substituted for one gramme of hydrogen Conversion from mg /100 ml is effected by the following formula

$$\text{mEq /l} = \frac{10 \times \text{mg /100 ml} \times \text{valency}}{\text{atomic weight}}$$

| ESTIMATION | WHOLE BLOOD | PLASMA OR SERUM |
|---|---|---|
| Alkali reserve (as $HCO_3$) | — | 53–77 vol $CO_2$% (24–25 mEq /L) |
| Amino acid nitrogen | 4–8 | 3–7 |
| Bilirubin (Van den Bergh) | — | 0 1–0 8 |
| Calcium (total) | 5–7 | 9–11 (4 5–5 5 mEq /L) |
| (diffusible) | — | 4 2–5 6 (2 1–2 8 mEq /L) |
| Chlorides (as Cl ) | 270–310 (76–87 mEq /L) | 340–370 (96–105 mEq /L) |
| Cholesterol (total) | 110–230 | 140–280 |
| Creatine | 2–8 | 0 2–0 8 |
| Creatinine | 0 5–2 0 | 0 5–2 5 |
| Diastase (amylase) | — | 50 150 glucose units |
| Icterus index | — | 1–6 units |
| Iron | — | 0 08–0 18 |
| Non protein nitrogen | 25–30 | 20–40 |
| Phosphatase (acid) | — | 1–3 King Armstrong units/100 ml |
| Phosphatase (alkaline) | — | 3–13 King Armstrong units/100 ml |
| Phosphate (inorganic as P) | 2 5–5 | 2–4 5 |
| Potassium (as K ) | 150–250 (38–64 mEq /L) | 17–22 (4 4–5 6 mEq /L) |
| Protein (total) | — | 5 5–8 G /100 ml |
| Albumin | — | 3 5–6 G /100 ml |
| Globulin | — | 1 5–3 G /100 ml |
| Fibrinogen | — | 0 2–0 4 G /100 ml |
| Prothrombin time | — | 14–16 seconds |
| concentration | — | 70%–140% |
| Sodium (as Na ) | 170–225 (74–98 mEq /L) | 320–345 (139–150 mEq /L) |
| Glucose (fasting) | 60–110 | 60–110 |
| Urea | 15–40 | 15–40 |
| Uric acid | 1–4 | 1–4 |
| Vitamin A | — | 0 02–0 05 (70–20 i u /100 ml ) |
| Vitamin C | 0 6–1 8 | 0 5–2 0 |

## Approximate Equivalents

### *Weights*

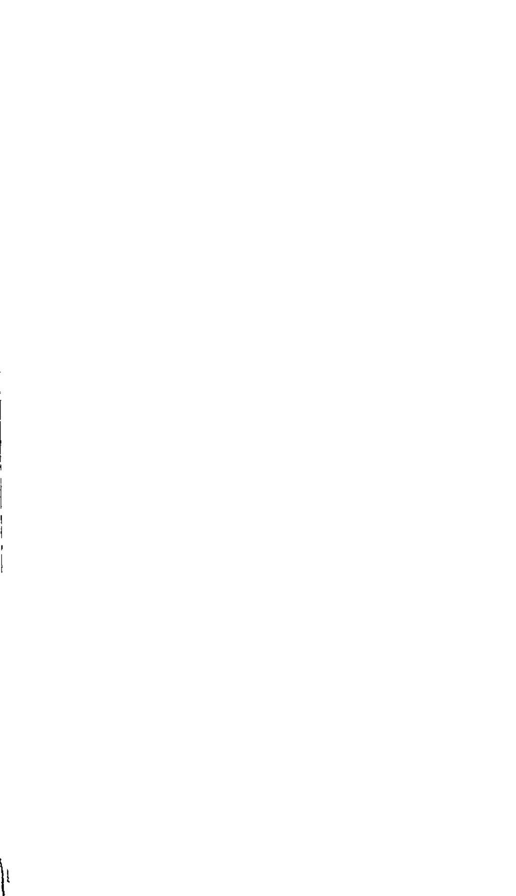
1 milligram (mg.) = 1/60 grain (gr.)
1 gramme (G.) = 15½ grains (gr.)
1 gramme (G.) = 1/30 ounce (oz.)
1 kilogram (kg.) = 2.2 pounds (lb.)
1 kilogram (kg.) = 1/6 stone (st.)

1 grain (gr.) = 60 milligrams (mg.)
1 ounce (oz.) = 8 drachms = 30 grammes (G.)
1 pound (lb.) = 16 ounces = 480 grammes (G.)
1 stone (st.) = 14 lbs = 6¼ kilograms (kg.)

### *Measures*

1 cubic centimetre (c.c. or ml.) = 17 minims (m.)
1 litre = 35 fluid ounces (fl. oz.)
1 litre = 1¾ pints

1 minim (m.) = 0.06 cubic centimetre (c.c. or ml.)
1 fluid drachm (fl. dr.) = 60 minims = 3.6 cubic centimetres (c.c. or ml.)
1 fluid ounce (fl. oz.) = 8 fluid drachms = 30 cubic centimetres (c.c. or ml.)
1 pint = 20 fluid ounces = 568 cubic centimetres (c.c. or ml.)



drugs such as mercury iodides bismuth or arsenic and it is also found in prolonged fevers and in cachectic conditions

**Clinical Findings** The patient complains of salivation a bad taste and a sensation of heat in the mouth

**Treatment** The cause should be removed if possible, and a mouth wash of pot permang 1 in 8 000 in water used frequently

### Aphthous Stomatitis
### (*Dyspeptic Ulceration*)

**Etiology** Aphthous stomatitis is often associated with debility gastro-intestinal disturbances and local infection with streptococci or staphylococci

**Clinical Findings** The patient is usually a woman There is pain in the mouth chiefly on mastication Small round or oval red areas are seen which rapidly ulcerate They vary in size from a pin s head to a split pea and usually occur inside the cheek under the tongue or on the gums They frequently recur

**Treatment** The ulcers should be touched with hydrogen peroxide 10 vols and a mouth wash of glycerin thymol co (B P C) diluted with 5 parts of warm water used In obstinate cases a silver nitrate stick may be applied to the ulcer but the possibility of inducing malignant disease by its repeated use must not be overlooked Penicillin lozenges 1 every hour for 8 doses are sometimes successful

### Ulcerative Stomatitis

**Etiology** Ulcerative stomatitis may be due to blood diseases such as acute leukæmia and agranulocytosis or to infection with Vincent s organisms In the latter case the ulceration is usually associated with debilitating diseases such as dysentery and scurvy in adults and measles and diphtheria in children

**Clinical Findings** The condition is the same as that described on p 1 the cheeks tongue and other parts of the mouth may be involved

**Treatment** This depends upon the cause and is described under the headings acute leukæmia agranulocytosis and ulcerative gingivitis

### Parasitic Stomatitis
### (*Thrush*)

**Etiology** Thrush is caused by the fungus *Oidium albicans*

**Clinical Findings** The patient is usually an infant or an adult who is very debilitated from diseases such as dysentery tuberculosis typhoid fever or cancer Whitish patches occur on the gums cheeks and tongue and may spread to the pharynx nose larynx œsophagus and stomach

**Treatment** Feeding bottles should be kept scrupulously clean and comforters forbidden Glycerinum boracis should be sparingly applied on gauze to the patches after each feed (boric acid poisoning may occur in an infant if excessive amounts are used) Alternatively a 1% aqueous solution of gentian violet may be applied on gauze t i d

The gums are painful and swollen Sloughing may occur and the teeth fall out The infection may spread to the tonsils or pharynx

Treatment The ulcers should be painted with a solution of liq arsenical m 90 tnc ipecac m 90 glycerin m 60 and in addition a mouth wash such as gargarisma pot chlorat should be used frequently Good results have been obtained in some cases by the oral administration of nicotinic acid tab mg 50 1 five times daily for 10 days A penicillin lozenge may be placed between the affected area of the gum and the cheek and allowed to dissolve every hour for 8 doses or the gum may be sprayed with a penicillin solution (1,000 units per ml ) every 2 hours In severe cases the best result is obtained by the intramuscular injection of 300 000 units of a procaine penicillin every morning and evening for 5 days

### Pyorrhœa Alveolaris

This may be a more advanced stage of marginal gingivitis or occur independently There is inflammation of the periodontal membrane around the tooth root and later rarefying osteitis of the alveolar margin No specific causative organism has been found but streptococci and various anaerobes are usually present Innumerable ills are attributed to pyorrhœa, from mild degrees of ill health to crippling attacks of rheumatism and fatal illnesses such as malignant endocarditis However this may be, pyorrhœa should be adequately treated by a dental surgeon but wholesale extractions for marginal gingivitis are to be deprecated

### Dental Abscesses

*The Alveolar Abscess* This may result either from dental caries leading to inflammation and death of the pulp of the tooth, or from periodontitis The abscess is painful

*The Small Apical Abscess* This is usually due to infection of the pulp in a crowned or carious tooth There is no actual pus present but a small granulomatous mass which often causes no local symptoms is revealed by X ray examination The focal infection is considered to be etiologically connected with numerous diseases, such as rheumatism thrombo phlebitis migrans infective endocarditis etc

The offending tooth should be extracted

### Halitosis

Definition Offensive breath

Etiology Halitosis may be due to numerous causes such as chronic tonsillitis pyorrhœa infection of the antra or adenoids, chronic gastritis bronchiectasis etc

### Stomatitis

Definition Inflammation of the mucous membrane lining the mouth Six varieties are described

### Catarrhal (Simple) Stomatitis

Etiology Catarrhal stomatitis may be due to local causes such as a sharp tooth very hot food or over smoking It may also result from

the anterior chamber of the eye It occurs chiefly in the Eastern Mediterranean area The eyes may be sore and painful and eventually blindness may ensue There is a tendency for spontaneous recovery and relapses In some instances the central nervous system is involved with headache vertigo and diplopia Antibiotics are of no avail In one of my cases a favourable result occurred after protein shock therapy 100 millions of T A B organisms being injected intravenously Cortisone or corticotropin may be tried the former in doses of mg 25 tab t.i d by mouth the latter mg 25 daily in an intravenous saline drip for 5 to 6 days

### Ludwig's Angina

This is an acute infection of the floor of the mouth due to the streptococcus There is a hard brawny swelling under the jaw and in the neck and the patient is very ill Treatment consists in free and deep incisions and the intramuscular injection of crystalline penicillin 500 000 units six hourly

### Tonsillitis

Definition Inflammation of the tonsils

The following varieties are described —Acute including acute follicular tonsillitis and Vincent s angina Chronic tonsillitis

### Acute Follicular Tonsillitis

Etiology There is infection of the tonsils with pyogenic organisms usually streptococci The condition may be primary, or develop in association with such diseases as rheumatic fever acute nephritis scarlet fever or secondary syphilis

Pathology The tonsils are swollen and an exudate collects in the follicles

Clinical Findings The patient is often a child or young adult who complains of malaise with pain in the throat made worse by swallowing

*On Examination* The temperature is usually raised to 101 F or more the tonsils are red enlarged and yellowish white spots may be seen on them due to the exudation in the follicles This exudation may coalesce and form a membrane which however is usually limited to the tonsils and does not spread to the pharynx or uvula The cervical glands are generally only slightly enlarged A swab should be examined to exclude the presence of diphtheria bacilli or the organisms of Vincent s angina

Differential Diagnosis It is important to exclude diphtheria Vincent s angina the onset of scarlet fever and the presence of active syphilis

Course and Complications The disease usually lasts 7 to 10 days Nephritis may occur as a complication and the urine should always be tested for protein and blood Infection may spread to the cranial sinuses or middle ear or the cervical glands may become chronically enlarged Septicaemia and toxic myocarditis are rare but important

for 3 or 4 days Treatment in adults is mainly that necessary for the debilitating condition

## Gangrenous Stomatitis

*(Cancrum Oris Noma)*

**Etiology** The treponeme and fusiform bacillus of Vincent are usually present

**Clinical Findings** The patient is usually a child who is very debilitated owing to measles diphtheria scarlet fever or whooping cough Pain is noticed in the mouth, and an ulcer is seen on the inner side of the cheek which may rapidly spread and perforate the cheek There is œdema of the face and the gums or jaw may be similarly affected The temperature is usually high and death frequently follows from toxæmia or bronchopneumonia

**Treatment** A course of three hourly intramuscular injections of crystalline penicillin should be given using 5 000 units for every year of the child s age

### *Vesicular Stomatitis*

**Etiology** This may be a manifestation of herpes when it is known as herpetic stomatitis or herpes buccalis The herpetic eruption may be limited to half of the hard palate or involve the tongue gums and cheek *on one or both sides There is malaise, and pain both inside and outside* the mouth especially on mastication The vesicles are situated on an inflamed base of mucous membrane

In foot and mouth disease which rarely affects man, vesicular stomatitis (epizootic stomatitis) may occur

**Treatment** A mouth wash of glycerin thymol co (B P C ) diluted with 5 parts of warm water should be used

## The Stevens Johnson Syndrome

*(Erythema exudativum multiforme bullosum with conjunctivitis and stomatitis)*

This syndrome is characterised by stomatitis conjunctivitis a bullous skin rash and fever In some cases there is urethritis and balanitis The cause is unknown It is most commonly met with in males under the age of 20 years Loss of vision may result from corneal ulceration or deeply seated ocular inflammation The rash tends to have a peripheral distribution and may be mistaken for small pox

**Treatment** Procaine penicillin 300 000 units should be injected intramuscularly morning and evening and sulphamerazine 1 G given by mouth every 4 hours for 7 to 10 days One per cent hydrocortisone eye drops 2 drops in each eye every 2 hours may be helpful together *with intramuscular injections of mg 25* ACTH (corticotropin) every 6 hours for 4 or 5 days the dose being then tapered off during the next week

## Behçet s Syndrome

*This* appears to be allied to the Stevens Johnson syndrome It is characterised by recurrent genital and oral ulceration with lesions of

squeezed out from the tonsils The adenoid tissue at the back of the nose may be hypertrophied The glands in the neck may be enlarged especially below the angle of the jaw If there is marked obstruction to respiration and the patient is a mouth breather he shows the characteristic appearances thus the lips are dry and cracked, the central teeth prominent the gums are dry, and marginal gingivitis may be present Further, the palate is often narrow with a high arch and the nose is thin with feebly developed alar nasal cartilages The chest may be pigeon breasted There is often some deafness restlessness at night and the child is backward at school

**Course and Complications** The patient is liable to colds sore throats, otitis media bronchitis and general ill health Local or systemic infections such as diphtheria scarlet fever rheumatism etc may occur in association with chronic tonsillar sepsis

**Prognosis** This is favourable as the disease can be eradicated surgically

**Treatment** In the majority of cases the tonsils and adenoids should be removed by operation Medical treatment consists in the use of a throat paint such as the Pigmentum Mandl (Iodin gr 6 pot iod gr 20 ol menth pip m 5 glycerin ad fl oz 1) or the administration of penicillin as lozenges and by injection as described above for acute tonsillitis

*The Lingual Tonsil* This is situated at the base of the tongue and when enlarged may cause a persistent irritating cough curable by operation

## Quinsy

(*Peritonsillar Suppuration Peritonsillitis*)

**Definition** Abscess formation in the connective tissue around the tonsil

**Etiology** Quinsy is generally secondary to chronic tonsillitis

**Clinical Findings** The patient who is usually a young adult feels ill with shooting pains in the ear and throat rigors often intense dysphagia and inability to open the mouth fully

*On Examination* The breath is offensive and the mouth is kept slightly open A swelling due to the abscess is seen usually above and on the outer side of one or other tonsil This pushes the tonsil out of view and the uvula is deviated from the mid line

**Differential Diagnosis** A sarcoma of the tonsil may be mistaken for quinsy with the former there is usually no fever

**Course and Complications** The abscess points in a few days and usually bursts through the anterior pillar of the fauces with immediate relief of pain Complications are rare They include suppuration in the neck pyæmia hæmorrhage œdema of the glottis thrombosis of the internal jugular vein or even asphyxia from inhalation of the pus if the abscess ruptures during sleep Blockage of a few bronchi by inhaled pus will lead to collapse of a portion of the lung and hæmoptysis may result from the inflamed bronchial mucous membrane

**Treatment** In the early stages the treatment is as for acute

complications Recurrences are common, especially in the early or late winter

**Prognosis** This is usually good Repeated attacks lead to small fibrosed and cryptic tonsils which may become foci of infection for quinsy or other diseases, such as rheumatism, sciatica, or endocarditis Death may occur from septicæmia or myocarditis

**Treatment** The patient should be put to bed kept on a liquid or semi solid diet and the bowels opened with an aperient Local treatment Hot gargles of potassium permanganate (1 in 8 000) should be used frequently if possible If the patient cannot gargle the throat may be sprayed with a hot alkaline lotion such as Sod bicarb sod benzoat sod chlorid āa gr 10 aq ad fl oz 1 fl oz ½ to be used in a tumblerful of warm water occasionally, or the throat may be painted with Salol gr 30 glycerin m 120 sp vin rect ad fl oz 1 By mouth a mixture can be given such as Sod salicyl gr 10 sod bicarb gr 20 pot chlorat gr 5 dextros gr 30 aq ad fl oz 1 Fl oz 1 t d s p c The majority of cases of acute tonsillitis can be rapidly cured by the intramuscular injection of 300 000 units of a procaine penicillin morning and evening for 4 or 5 days If recurrences are frequent tonsillectomy is usually advisable

### Vincent's Angina

**Etiology** Ulceration of the tonsil due to infection with the B fusiformis and the Treponema vincenti

**Clinical Findings** The patient complains of symptoms resembling *those of subacute tonsillitis There is not usually severe pain and the* temperature is normal or slightly raised In some cases the patient is severely ill with high fever and muscle and joint pains

*On Examination* A yellowish white membrane or ulcer may be seen on or behind one tonsil A swab should be examined for the presence of diphtheria bacilli and Vincent s organisms

**Differential Diagnosis** Acute tonsillitis diphtheria rheumatic fever, syphilis and agranulocytic angina must be excluded

**Course** This is benign the condition usually clearing up spontaneously in about a week but some cases prove more intractable

**Treatment** The throat should be sprayed every 2 hours with a penicillin solution containing 1 000 units per ml and a course of intra muscular injections of 300 000 units of a procaine penicillin given morning and evening for 5 to 7 days

### Chronic Tonsillitis

**Etiology** Chronic tonsillitis may result from acute tonsillitis or develop insidiously

**Clinical Findings** The patient is usually a child or young adult who gives a history of repeated attacks of sore throat generally in the winter

*On Examination* The tonsils usually look unhealthy ' being some times large and almost meeting in the mid line or else of normal size but showing depressions or pits or small and scarred Pus or cheesy exudate of very offensive odour may in some cases be

condition and is of no pathological significance In tertiary syphilis the tongue may be fissured

*A white strawberry tongue* occurs in scarlet fever The papillæ are covered with a white fur

*A red strawberry or raspberry tongue* occurs in a later stage of scarlet fever The tongue has peeled and the bright red papillæ stand out

*A dry glazed or brown tongue* occurs in the terminal stages of severe illnesses such as cholera dysentery and septicæmia

*A slaty blue tongue* may be seen in Addison's disease

*A black tongue* (melanoglossia) The black tongue is usually hairy due to overgrowth of filiform papillæ on the centre of the dorsal surface The colour is usually thought to be due to a fungus, the Aspergillus niger whose spores are black In other cases yeasts or bacteria producing black colonies have been isolated but the black colour may be due to food debris or the papillæ may darken with age Other causes which have been suggested include chronic naso pharyngeal infection tobacco smoke irritant mouth washes sulphonamide drugs penicillin chloramphenicol aureomycin and terramycin gastric hyper secretion and a trophoneurosis It is not due to nicotinic acid deficiency which causes black tongue in dogs It usually causes no ill effects and may disappear spontaneously treatment being not likely to prove efficacious The tongue should be scraped daily and attention paid to oral and dental hygiene The patient should not smoke The application of a 1 in 1 000 solution of thymol has been recommended for fungus infections

*A sore tongue* may occur in ulceration from any cause in the early stages of pernicious anæmia in sprue or in mercurial poisoning

*A smooth glazed tongue* may be seen in pernicious or simple achlorhydric anæmia

## Glossitis

**Definition** Inflammation of the tongue There are two varieties acute and chronic glossitis

### Acute Glossitis

**Etiology** Acute glossitis may result from abrasions of the tongue associated with dental caries or from insect bites or burns Occasionally acute glossitis complicates severe fevers or small pox or results from sucking penicillin lozenges The infection is usually streptococcal

**Clinical Findings** The patient complains of pain and swelling of the tongue and the pain may radiate to the ears There is usually marked prostration and high fever

*On Examination* The tongue is red and swollen and enlarged glands may be felt in the neck

**Course and Complications** The disease is usually rapidly progressive Abscesses or gangrene may occur in the tongue and complications include œdema of the larynx Ludwig s angina and septicæmia

**Prognosis** This is very unfavourable the majority of cases proving fatal

follicular tonsillitis The attack is best terminated surgically by incision as soon as pus has formed, and the tonsils should be removed later

## Tuberculosis of the Tonsils

**Pathology** The tonsil may very rarely be affected clinically apart from pulmonary tuberculosis The patient complains of a chronic sore throat and generally of symptoms of pulmonary tuberculosis such as cough expectoration, malaise etc

*On Examination* The tonsil may be ulcerated In the majority of cases there is definite pulmonary and laryngeal tuberculosis and tubercle bacilli are present in the sputum In another group of cases the clinical features are those of cervical adenitis the tubercle bacilli gaining entrance through the tonsils and settling in the glands in the neck In such cases tubercles may or may not be present macroscopically in the tonsils but tonsillectomy is usually advisable In all cases a course of streptomycin para aminosalicylic acid and/or isoniazid should be given (see p 168)

## Syphilis of the Tonsils

The tonsil may be infected in the primary stage when the lesion present is a chancre In secondary syphilis a sore throat with acute tonsillitis or the formation of superficial plaques (snail track ulceration) on the tonsils may occur In tertiary syphilis ulceration due to a gumma may be noted

## Tumours of the Tonsil

These are nearly always malignant, being either epitheliomatous or sarcomatous (lymphosarcoma or round celled sarcoma) In epithelioma there is usually ulceration of the tonsil and hard enlarged glands in the neck In lymphosarcoma the tonsil appears swollen pale and waxen or may be ulcerated It grows rapidly and neighbouring glands are usually soon enlarged

## The Tongue

An examination of the tongue does not give so much information concerning the health of the patient as was at one time believed Thus the tongue may be almost black (melanoglossia) apart from taking drugs and yet the patient be in good health or with a tongue clean and moist the patient may be suffering from a mortal illness such as tuberculosis or cancer

*Furred tongue* A white or brown coat may result from a milk diet or from drugs such as bismuth or iron or it may be associated with fevers chronic alcoholism gastro intestinal disorders or local causes such as carious teeth or heavy smoking

*A soft flabby tongue* with impressions of the teeth on its edges may occur in atonic dyspepsia and gastric hyposecretion

*A red firm tongue* may be associated with gastric hypersecretion or with diabetes mellitus (raw beef tongue)

*A fissured tongue* (scrotal tongue) occurs usually as a congenital

forming outlines resembling a map Fresh lesions begin as small whitish patches which shed their epithelium at the centre

**Course** The condition generally persists for some time

**Treatment** Irritants should be avoided and a simple mouth wash of half a teaspoonful of sod bicarb in a glass of warm water used morning and evening

### Ulcers of the Tongue

These may be 1 Simple, due usually to irritation of a tooth "dyspeptic, or associated with ulcerative stomatitis Sub-lingual ulcers may be met with in whooping cough in cretinism associated with a large tongue or in ravenous breast fed infants 2 Granulomatous caused by syphilis or tuberculosis 3 Malignant an epithelioma

### Tumours of the Tongue

These are simple or malignant A papilloma may occur sessile or pedunculated Operative removal is always advisable An epithelioma is usually found in men over the age of 40 It may develop from a papilloma or from a leukoplakic patch There is a tendency to ulceration and secondary deposits are found in the cervical glands Treatment is surgical either by radium, diathermy or excision

### Granulomata of the Tongue

A gumma forms a firm slightly raised swelling often seated near the centre of the tongue

Tuberculosis causes painful ulceration frequently near the tip and is associated generally with pulmonary tuberculosis

## THE PHARYNX

### Pharyngitis

**Definition** Inflammation of the pharyngeal mucous membrane

There are two varieties Acute and chronic Acute pharyngitis may be catarrhal or septic

### Acute Catarrhal Pharyngitis

*(Sore Throat)*

**Etiology** Acute catarrhal pharyngitis may be due to a cold or occur at the onset of specific fevers such as measles or scarlet fever or *be caused by drugs, such as iodides* or *mercury* *It* is also sometimes seen in the secondary stage of syphilis or it may follow tonsillitis

**Clinical Findings** The patient complains of rawness at the back of the throat with perhaps dysphagia

*On Examination* The pharynx is red and congested General constitutional disturbance with slight pyrexia is usually present

**Course and Complications** A mild form of laryngitis may follow after the pharyngitis has disappeared

**Prognosis** This is usually good

**Treatment** Relief is obtained by a steam inhalation containing

**Treatment** Cold applications such as ice should be placed on the tongue Penicillin should be administered by injection as described above for acute tonsillitis

### Chronic Glossitis

**Etiology** Chronic glossitis may result from irritation due to smoking from syphilis chronic alcoholism or occur in association with oral sepsis anæmia and achlorhydria A raw glazed and superficially fissured tongue often associated with cracked leukoplakic angles of the mouth (angular stomatitis) may be met with in pellagra tropical sprue, pernicious anæmia, the nutritional anæmias and idiopathic steatorrhœa It is thought to be due to deficiency of the vitamin B complex

**Clinical Findings** The patient complains of a sore tongue and in severe cases the pain may keep him awake

*On Examination* Red smooth patches may be seen on the tongue

**Course and Complications** The condition is usually of long duration and leukoplakia may develop

**Prognosis** This is usually good if adequate treatment is begun early

**Treatment** All irritating food should be avoided smoking and alcohol must be forbidden Any septic focus in the mouth or a sharp tooth should be treated Tincture of hamamelis m 60 in water fl oz 1 may be applied to the tongue night and morning Nicotinic acid tab mg 50 1 or 2 t i d should be given by mouth for the glossitis due to vitamin B deficiency and for the angular stomatitis riboflavin tab mg 10 t d s

### Leukoplakia Buccalis

**Definition** A condition of keratosis of the tongue or mucous membrane of the mouth with thickening of the deeper tissues

**Etiology** Leukoplakia is usually associated with syphilis over smoking chronic alcoholism and oral sepsis

**Clinical Findings** The patient is commonly a male over the age of 40 There are usually no symptoms, but thickened whitish grey patches occur on the tongue, and at times on the cheeks or gums

**Course and Complications** Ulceration or epitheliomatous changes are prone to occur

**Treatment** Owing to the risk of malignant disease excision of the patches is usually recommended Anti syphilitic treatment is generally of no avail

### Geographical Tongue

(*Eczema of the Tongue*)

**Etiology** The cause is unknown

**Clinical Findings** The patient may complain of itching or burning of the tongue

*On Examination* Whitish rings may be seen surrounding a red area of the tongue The rings may spread and fuse with each other

night and morning Septic foci in the mouth and nose should be treated speakers should rest the voice and if necessary take lessons in voice production

## Retropharyngeal Abscess

**Definition** Suppuration in the submucous connective tissue behind the posterior pharyngeal wall The prevertebral glands are first affected

There are two varieties Acute and chronic

### Acute Retropharyngeal Abscess

**Etiology** Acute retropharyngeal abscess may occur in association with suppuration in the nose or posterior pharyngeal lymph glands septic tonsillitis otitis media or as a complication of scarlet fever diphtheria and measles

**Clinical Findings** The patient is usually an infant who has pain in the throat and difficulty in swallowing or breathing The cry may be quacking in character (*cri de canard*) and croup sometimes occurs

*On Examination* The child appears ill and is feverish A bulging of the posterior pharyngeal wall can be seen usually not quite central It may be possible to feel fluctuation

**Course** Death may occur from rupture of the abscess and suffocation

**Treatment** This is surgical An incision should be made through the mouth into the posterior pharyngeal wall without an anæsthetic the child s head being lowered so that the pus is not inhaled

### Chronic Retropharyngeal Abscess

**Etiology** The condition is usually tuberculous secondary either to tuberculous cervical or retropharyngeal glands or to caries of the upper cervical vertebræ

**Clinical Findings** The patient is usually a child, and is commonly afebrile There may be some dyspnœa or snoring but often there is no pain Enlarged tuberculous glands or signs of cervical caries may be evident and bulging may be seen in the posterior pharyngeal wall

**Treatment** This is surgical The abscess should be opened externally in the neck

## Pharyngeal Ulcers

These may be 1 Follicular 2 Syphilitic In primary syphilis a chancre may be seen in secondary syphilis there are mucous plaques or pharyngitis and in the tertiary stage ulcers or gummata may occur 3 Tuberculous Lupus may spread from the nose to the mouth palate and pharynx it shows a typical apple jelly appearance In advanced pulmonary tuberculosis there may be very painful ulcers in the pharynx 4 Accompanying fevers such as typhoid 5 Epitheliomatous 6 Diphtheritic

carbolic acid m 5 in one pint of steaming water at 160° F, or by a gargle of sod bicarb gr 15, aq ad fl oz 10

### *Acute Septic Pharyngitis*

**Etiology** Acute septic pharyngitis is due to a streptococcal or a pneumococcal infection

**Clinical Findings** The pharynx is red, œdematous or sloughing or a grey slimy exudate may form The patient is very ill with a subnormal or a high temperature

**Complications** These include œdema of the larynx Ludwig s angina pneumonia parotitis and septicæmia

**Prognosis** This is grave

**Treatment** Steam inhalations and hot fomentations may be used The throat may be sprayed with a penicillin solution containing 1 000 units per ml, and a course of 600 000 units of a procaine penicillin given intramuscularly every 12 hours for 5 to 7 days

### Chronic Pharyngitis

There are three varieties of chronic pharyngitis Simple or catarrhal (relaxed throat) granular or hypertrophic atrophic (pharyngitis sicca)

### Simple or Catarrhal Pharyngitis

Simple pharyngitis is due to over smoking chronic alcoholism dust or oral sepsis

**Clinical Findings** The patient is usually an adult male who com plains of rawness a tickling or pricking sensation in the throat, or of an ineffective paroxysmal cough The voice may be hoarse

*On Examination* The pharyngeal mucous membrane appears con gested and the venules may be dilated

### *Granular Pharyngitis*

Granular pharyngitis is also known as Clergyman s sore throat and may occur in association with catarrhal infections or some weakness of the voice and in children in association with adenoids

**Clinical Findings** The symptoms are much the same as those of simple pharyngitis

*On Examination* Small gelatinous looking swellings (lymphoid nodules) are seen on the pharyngeal wall

### Atrophic Pharyngitis

This is associated with rhinitis sicca

**Clinical Findings** The symptoms resemble those of simple or granular pharyngitis

*On Examination* The pharynx is red and shiny and muco pus may be seen *running down from the posterior nares*

**Treatment** In all forms of chronic pharyngitis irritants such as tobacco and alcohol should be avoided A throat paint of Liq ferri perchlor m 60 *glycerin ad fl oz 1 or Mandl s paint Iodin gr 6 pot* iod gr 20 ol menth pip m 5 glycerin ad fl oz 1 should be applied

**Treatment** Local or general causes should be treated Acid substances such as unsweetened lime juice and a mouth wash of glycerin and lemon juice may be useful

### Acute Septic Parotitis

### (*Parotid bubo*)

**Definition** Acute inflammation of the parotid gland

**Etiology** Acute septic parotitis is usually due to infection ascending the parotid duct The causative organism is frequently the staphylococcus aureus less often the streptococcus viridans or the Diplococcus pneumoniæ (pneumococcus) It may occur in the absence of mastication in fevers such as enteric in pneumonia in facial paralysis in cleft palate where an obturator is worn in chronic uræmia, after operations on the abdomen in treatment of peptic ulcer if a mouth wash is not used regularly after the feeds or in Sjogren s disease

**Clinical Findings** The patient complains of pain and swelling in the region of one parotid gland with dysphagia and malaise

*On Examination* A tender parotid swelling is found with redness of the overlying skin The patient is obviously ill

**Course and Complications** Suppuration usually occurs in the gland

**Prognosis** This is grave and death is not infrequent

**Treatment** The gland should be opened surgically as soon as fluctuation occurs previously fomentations may be applied Mouth washes should be used frequently If the infecting organism is penicillin sensitive a course of intramuscular injections of 600 000 units of a procaine penicillin should be given every 12 hours for 5 to 7 days

### Acute Specific Parotitis (see Mumps, p 575)

### Chronic Parotitis

In chronic inflammation of the parotid glands the other salivary glands are usually also affected This may result from such causes as drugs (iodides mercury or lead) syphilis mumps calculi and chronic nephritis Treatment is directed to the underlying cause

### Mikulicz s Syndrome

**Definition** A syndrome characterised by chronic swelling of the salivary and lachrymal glands

**Etiology** The cause of Mikulicz s syndrome is in some cases a low grade infection Some authorities consider that Mikulicz s syndrome and Sjogren s disease are the same

**Pathology** Various types occur varying from simple inflammation of the glands to leukæmia lymphadenoma and lymphosarcoma Syphilitic or tuberculous changes are rarely found in the glands

**Clinical Findings** The patient complains of swellings gradually increasing in size in the region of the eyes and face There is interference with vision, with some limitation of the temporal fields owing to the enlarged lachrymal glands dryness of the eyes and mouth but usually no pain

### Enlargement of the Uvula

The uvula may be enlarged in patients suffering from pharyngitis relaxed throat nephritis with œdema, or anæmia (œdematous) It may give rise to a cough

### Tumours of the Pharynx

These may be simple, such as a papilloma or angioma, or malignant such as an epithelioma or sarcoma Malignant tumours are rare

### Pharyngeal Neuroses

1 *Globus Hystericus* This is a motor spasm The patient complains of a lump in the throat The lingual tonsil may be enlarged

2 *Anæsthesia* This occurs especially in hysteria

3 *Paræsthesia* The patient complains of a feeling of suffocation and tickling in the throat

Diverticula of the pharynx are considered later

## THE SALIVARY GLANDS

### Ptyalism

### (*Salivation*)

**Definition** Over secretion of the salivary glands

**Etiology** The most important causes are 1 Drugs especially mercury iodides, arsenic and pilocarpine 2 Reflex Oral such as dental caries stomatitis and dentition Œsophageal as with a growth Gastric, at the onset of vomiting gastric ulcer etc Hepatic and pancreatic inflammatory lesions 3 Nervous lesions as in tabetic crises, tic douloureux encephalitis lethargica and paralysis agitans 4 Dysphagia due to mechanical causes as in fractured jaw mumps, etc or due to neuro muscular causes as in bulbar paralysis bilateral facial paralysis myasthenia gravis and hydrophobia In cases associated with dysphagia the saliva dribbles away and the secretion may not be excessive Ptyalorrhœa is a functional condition which may complicate pregnancy or occur in association with a high blood pressure

**Treatment** In each case this is directed to the underlying cause but atropine and bromides may be given Atropin sulph gr $\frac{1}{200}$, aq ad m 60 t d s a c or Pot brom gr 10 aq chlorof ad fl oz $\frac{1}{2}$ Fl oz $\frac{1}{2}$ t d s p c X ray treatment to the salivary glands may be required in obstinate cases

### Xerostomia

### (*Aptyalism*)

**Definition** Dryness of the mouth

*Etiology* Xerostomia may be due to Drugs especially belladonna and opium Fevers Deficient fluid intake Emotions such as fear Local causes such as thrush or chronic inflammation of the salivary glands as in Mikulicz s disease or in Sjogren s disease Old age especially in women In association with excessive loss of body fluids as in cholera and diabetes

of the mucous membrane to abscess formation Mediastinitis is often present

**Clinical Findings** There may be a history of any of the causative conditions described above The patient complains of pain on swallowing and under the sternum and there may be vomiting of blood and mucus In severe cases there is fever with rigors and marked constitutional disturbance

**Differential Diagnosis** The history of the case and the dysphagia with retrosternal pain usually render the diagnosis clear

**Course and Complications** In mild cases the patient rapidly recovers Perforation of the œsophagus or submucous abscess formation (phlegmonous œsophagitis) may lead to mediastinitis which is quickly fatal Lesser degrees of ulceration may result in subsequent stenosis

**Prognosis** This varies with the cause and the degree of inflammation There may be complete recovery death or subsequent stenosis

**Treatment.** No food must be given by mouth until the patient is able to swallow a little olive oil without pain Injections of morphin sulph gr $\frac{1}{8}$ to $\frac{1}{4}$ may be given during the acute stage The possibility of subsequent stenosis should be remembered Œsophagoscopy or an opaque meal will indicate any degree of obstruction with dilatation above it Stricture should be prevented by the passage of bougies

## Chronic Œsophagitis

**Etiology** Chronic œsophagitis may be due to alcoholism achalasia of the cardia hiatus hernia frequent vomiting associated with pyloric stenosis repeated passage of a stomach tube tuberculosis syphilis actinomycosis diverticula new growths or a cerebral tumour

**Clinical Findings** The symptoms of chronic œsophagitis are usually indistinguishable from those of the underlying causes which are described later In addition there is usually a burning sensation in the back and under the lower third of the sternum Repeated small hæmatemeses may occur and œsophageal spasm will cause dysphagia

**Treatment** The diet should be soft and bland and the following mixture given Sod brom gr 10 tinc belladon m 15 aq chlorof ad fl oz 1 Fl oz 1 t d s In addition olive oil m 60 to 120 should be given immediately before each feed

## Œsophageal Obstruction

**Etiology** The blockage may be due to 1 Obstruction in the lumen as by impacted foreign bodies 2 Changes in the walls (intrinsic causes) such as stenosis congenital or acquired the latter may be due to spasm Other causes include fibrosis from ulceration (due to trauma chemicals diphtheria new growth or gumma) and tumours especially carcinoma 3 External pressure (extrinsic causes) due to aneurysm an enlarged thyroid enlarged glands mediastinal growth pericardial or pleural effusion pharyngeal or œsophageal diverticula vertebral exostoses or new growths or cervical caries

Certain of these conditions will now be described in more detail

*On Examination* Swellings are seen in the region of the lachrymal parotid and submaxillary glands and there may be ptosis of the eyelids

**Differential Diagnosis** The gradual onset absence of pain and the disposition of the swellings usually make the diagnosis clear The uveoparotid syndrome of Heerfordt may be mistaken for Mikulicz s syndrome Heerfordt's syndrome is a manifestation of sarcoidosis and there is enlargement of the parotid submaxillary, and sometimes of the lachrymal glands with inflammatory lesions in the uveal tract and often facial paralysis or more rarely polyneuritis In Sjogren s disease there is dryness of the eyes mouth nose pharynx larynx and bronchi in addition to salivary gland enlargement There may also be a polyarthritis

**Course and Complications** The course is usually chronic Leukæmic or lymphosarcomatous changes may show themselves

**Treatment** Arsenic and iodides may be given by mouth such as Liq arsenical m 2 pot iod gr 5 sod bicarb gr 10 sp chlorof m 5 infus gent co rec ad fl oz 1 Fl oz 1 t d s p c This usually has no effect Cortisone (mg 25 tab ) in doses of mg 75 to 100 daily by mouth may be tried for a week The glands may be treated by X rays or surgical removal of the enlarged lachrymal glands may be followed by disappearance of the swelling of the salivary glands

### Tumours of the Salivary Glands

The *majority* of tumours of the *salivary glands* are *mixed* ones containing myxomatous fibrous and cartilaginous tissue with a low grade of malignancy They should be removed surgically and the scar afterwards treated with X rays The parotid gland is usually affected *Localised fatty infiltration* of the parotid simulating a mixed parotid tumour, has been described

### Salivary Calculi

Calculi rarely bilateral *form usually in the submaxillary glands or* ducts They may give rise to painful swelling of the glands especially provoked by foods which stimulate salivation or by acid substances The calculus may be palpable or may only be seen by X rays It should *be removed surgically*

## THE ŒSOPHAGUS

### Œsophagitis

***Definition*** *Inflammation of the œsophagus* *This may be acute* or chronic

### Acute Œsophagitis

**Etiology** Acute œsophagitis may be due to chemical irritants such as poisons (carbolic acid caustic soda perchloride of mercury or poison gases) to impacted foreign bodies or it may be associated with tuberculosis diphtheria syphilis or carcinoma It may also occur in small pox or enteric fever

**Pathology** *In cases of poisoning the lesion is usually situated at* the lower end of the œsophagus The changes ary from hyperæmia

## Cardiospasm

*(Achalasia of the cardia   Idiopathic dilatation of the œsophagus   Phrenospasm   Hiatal œsophagismus)*

**Etiology** Obstruction occurs at the lower end of the œsophagus due to failure of relaxation of the cardiac sphincter on deglutition and caused probably by a disturbance of the neuro muscular mechanism There is probably no spasm of the right crus of the diaphragm The cardiac sphincter consists of circular fibres 1 inch in depth and situated 1 to 2 inches above the œsophago gastric junction Vagal stimulation causes contraction of the longitudinal fibres of the œsophagus and relaxation of the cardiac sphincters Sympathetic stimulation has the reverse effect Achalasia of the œsophagus may be due to fracture of the base of the posterior fossa of the skull the last four cranial nerves being involved either by a hæmatoma or by the fracture passing through the jugular foramen and hypoglossal canal This is known as Collett s syndrome A case has been described in which there was complete achalasia at both the upper and lower ends of the œsophagus associated with a fractured skull and hæmatoma There may be a psychogenic element in cases of achalasia associated with emotion or grief

**Pathology** The œsophagus is dilated and it may be lengthened and curved There is inflammation of the mucous membrane and ulcers may form at the lower end of the œsophagus The cardia is normal There is degeneration of Auerbach s plexus in the cardiac sphincter in some cases The œsophagus may hold as much as 3 pints instead of the normal 3 oz and it may measure 12 inches in diameter

**Clinical Findings** The patient is usually an adult over the age of 20 of either sex He may give a history of discomfort on swallowing for many years and later of regurgitation of solid food or at times of a feeling of obstruction The patient may prefer to take his meals standing up There is also often a choking sensation and sub sternal pain This occurs usually directly after swallowing but when much dilatation has taken place vomiting may be delayed for half an hour or so Œsophageal vomiting may occur during sleep Achalasia of the cardia may ensue directly after the vomiting of pregnancy Pressure symptoms may occur such as dyspnœa palpitations and pain extending to the shoulders

*On Examination* The patient is not usually wasted except in advanced cases and no physical signs are found The opaque meal shows the dilated œsophagus above and obstruction at the level of the diaphragm

**Differential Diagnosis** Other causes of œsophageal obstruction must be excluded especially carcinoma of the œsophagus a fibrous stricture resulting from a healed ulcer and aneurysm The barium meal and X ray examination may suggest the irregular outline of a carcinoma and a fractional test meal may show a curve suggestive of carcinoma of the stomach Thus there may be high total acidity no free HCl and some blood if the fermenting contents are aspirated from the dilated œsophagus Clinically the long history and absence of

### Congenital Atresia

The œsophagus ends in communication with the trachea or a bronchus, so that food swallowed passes into the lungs causing cough and regurgitation. Less frequently the upper portion of the œsophagus ends blindly, the lower end communicating above with the trachea or a bronchus. Excessive mucus in the pharynx is usually noted at birth. Death usually occurs a day or so after birth.

### Congenital Stenosis

The lumen of the œsophagus is narrowed but there is no cicatrisation. Clinically, regurgitation of food shows itself when the infant takes solids.

### Œsophageal Spasm

(*Œsophagismus*)

Three varieties occur —

1 *Primary Œsophageal Spasm* This is a neurosis The patient experiences difficulty in swallowing the bolus sticking in the gullet Globus hystericus, hiccough or regurgitation of food may occur The spasm is transitory and may take place at any level in the œsophagus

**Treatment** Cure can usually be effected by suggestion

2 *Reflex Œsophageal Spasm* This may occur in association with local lesions such as a growth or ulcer in the œsophagus or with inflammation of the gall bladder or stomach

3 *Plummer Vinson Syndrome* This is also known as the Paterson Kelly syndrome or sideropenic dysphagia It is probably caused by a disturbance of the neuro muscular mechanism at the junction of the pharynx and œsophagus

**Pathology** There is absence of relaxation of the pharyngo œsophageal sphincter

**Clinical Findings** The patient is often a woman, about the age of 40 There is difficulty in swallowing solids and in some cases liquids, the food sticking at the back of the throat

*On Examination* The tongue is smooth and may be sore The pharynx is pale and dry The skin is pale brownish yellow the nails are spoon shaped (koilonychia) and there are cracks at the corners of the mouth (perlèche) The blood shows a microcytic hypochromic anæmia with increased fragility of the red cells It has been recorded with a normal blood count but a low serum iron level Achlorhydria is frequently found A barium swallow X ray examination may show a post cricoid web This is a small fold of mucous membrane projecting into the lumen of the œsophagus which can also be seen on œsophago scopy The spleen and liver may be enlarged

**Course and Complications** The course is progressive if untreated Carcinoma at the junction of the pharynx and œsophagus (post cricoid carcinoma) and pernicious anæmia may occur as complications

**Treatment** This consists in the passage of a bougie and the administration of ferri et ammon cit gr 20 to 40 t d s p c (see p 518) or if this fails iron should be injected intravenously or intramuscularly (see p 518)

Œsophagoscopy will usually confirm the diagnosis Blood may be present in the vomit or in the stools Enlarged glands may be felt in the neck, especially above the left clavicle

**Differential Diagnosis** Other causes of œsophageal obstruction, such as aneurysm, achalasia and syphilitic stricture must be excluded as described above

**Course and Complications** The course is progressive Complications such as perforation of the œsophagus hæmorrhage and extension of the growth into the surrounding structures such as the mediastinum and lungs and septic bronchopneumonia may occur The involvement of the mediastinum may give rise to deficient air entry into one or other lung or to unilateral or bilateral recurrent laryngeal paralysis

**Prognosis** Death usually occurs within 6 to 12 months from the onset of symptoms

**Treatment** This is largely palliative an early gastrostomy may prolong life by enabling nutrition to be maintained by direct feeding into the stomach To relieve dysphagia Souttar s tube may be used This is ' a flexible spiral formed of German silver wire and gilded It has an expanded upper end and a twisted oval section which prevents displacement It is introduced through the growth with an œsophagoscope and allows the patient to swallow solid food Radium may be inserted into the œsophagus or the growth treated with a radium bomb or by deep X rays Total excision is an operation with a high mortality rate but a few successful cases have been recorded

### Sarcoma of the Œsophagus

This is much less common than carcinoma It may form a polypoid or ulcerating growth

### Simple Tumours of the Œsophagus

Simple polypi may occur giving rise to obstructive symptoms Œsophagoscopy enables them to be diagnosed and removed Other simple tumours include fibroma fibromyoma an accessory thyroid tumour and a simple cyst

### Syphilis of the Œsophagus

**Etiology** Syphilis of the œsophagus is usually due to acquired disease rarely occurring in the congenital variety

**Pathology** In secondary syphilis there is inflammation of the mucous membrane The lesions take the form of an œsophagitis which may give rise to dysphagia Gummata may occur in tertiary syphilis at the upper or lower end of the œsophagus These may ulcerate Leukoplakia is often seen in the mouth and in the œsophagus Obstruction is intensified by muscular spasm

**Clinical Findings** The patient is usually an adult of either sex who complains of progressive dysphagia There is commonly no pain unless the pharynx is also involved Signs of syphilis may be found

*marked cachexia* and wasting negative this diagnosis and œsophago scopy excludes it

**Course and Complications** Remissions often occur in the early stages but usually the condition *becomes worse if untreated* *Hæmor* rhage, the formation of a diverticulum rupture of the œsophagus and general wasting may ensue Carcinoma is a very rare complication

**Prognosis** This is favourable if proper treatment is given especially if the condition has not existed very long

**Treatment** There is little evidence that psychiatric treatment is ever effective The food should be soft and well chewed In some cases the inhalation of 8 m of octyl nitrite from a crushable glass capsule or from an inhaler, affords relief and even effects a cure The drug may be inhaled before directly after or during a meal and this treatment is *worthy of trial in any case If this fails* a mercury bougie *should* be passed at first directly before every meal and later at less frequent intervals The bougie is a closed rubber tube about ½ inch in diameter, containing mercury and weighing about 1 lb 5 oz The weight of the tube causes its passage after swallowing down the œsophagus and into the stomach The patient soon learns to do this for himself and to begin with the bougie should be left *in situ* for 10 to 15 minutes each time it is passed Later it is removed as soon as it has been passed Many surgeons consider the mercury bougie should not be used Surgical treatment consists in dilatation of the sphincter by a solid bougie, *rupture of the circular sphincter muscle fibres by a Negus* dilator or Heller s operation in which the circular muscle fibres at the lower end of the œsophagus are divided down to the mucous membrane In other cases a lower œsophageal sympathectomy has been success ful

## Carcinoma of the Œsophagus

**Etiology** The cause is unknown It may be associated with food irritants

**Pathology** The growth usually occurs in the upper or lower end of the œsophagus *or where it is crossed by the left bronchus Three* types occur—an ulcerative a scirrhous or more rarely a fungating form It may infiltrate surrounding structures in the neck or mediastinum The growth is usually of the squamous celled variety but the fungating form may be a columnar celled adenocarcinoma arising from the mucous glands

**Clinical Findings** The patient is usually a male over the age of 40 He complains of dysphagia which may have a *sudden onset* but more often comes on gradually being first noticed on swallowing solids and later fluids Subsequently vomiting occurs directly after swallowing often of frothy material mixed with food An early symptom may be substernal pain The patient rapidly loses weight and becomes cachectic as the stenosis increases

*On Examination* In the early stages there are no physical signs An X ray examination with the screen and swallowed *barium meal* indicates the point of stricture and excludes the presence of an aneurysm

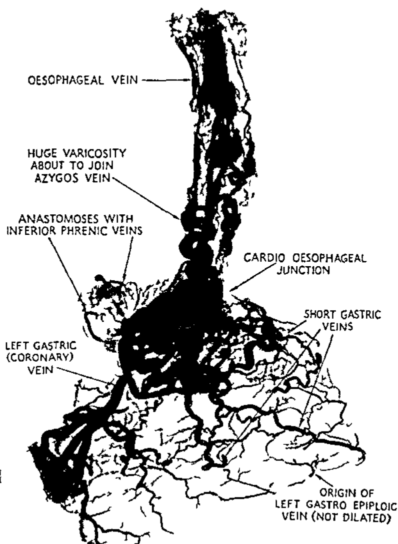



FIG. 1 OESOPHAGEAL AND GASTRIC VARICES PHOTOGRAPHED AFTER INJECTION WITH BISMUTH

elsewhere The blood Wassermann reaction may be negative, but that of the cerebro spinal fluid is usually positive

**Differential Diagnosis** This can only be made with certainty by œsophagoscopy as a positive Wassermann reaction due to syphilis elsewhere may occur in association with a carcinoma of the œsophagus

**Course and Complications** Progressive stenosis usually develops if the lesion is left untreated

**Prognosis** This is very favourable with adequate treatment

**Treatment** A full course of anti syphilitic treatment should be given (see p 596) Stenosis should be prevented by repeated dilatation with bougies

### Diverticula of the Œsophagus

**Definition** Pouches formed by herniation of the œsophageal mucous membrane through the muscular coat

**Etiology** There are two varieties 1 *Traction Diverticula* These are caused by adhesions between the œsophagus and chronically inflamed tuberculous bronchial glands at the bifurcation of the trachea 2 *Pulsion Diverticula* These are due to increased internal pressure associated with swallowing food and weakness of the œsophageal wall

**Pathology** Traction diverticula are usually small and the whole coat of the œsophagus is involved They arise from the anterior wall near the tracheal bifurcation Pulsion diverticula may be small or large The pharyngo-œsophageal (pharyngeal) diverticulum arises from the posterior wall of the pharynx at the weak spot just above the crico pharyngeal muscle The opening is immediately above the superior opening of the œsophagus The diverticulum usually passes to the left and thence forward Small pulsion diverticula may arise laterally in the lower part of the œsophagus

**Clinical Findings** Diverticula usually give rise to no symptoms unless they are sufficiently large to interfere with deglutition Accumulations of small quantities of food may cause an unpleasant taste regurgitation of food may occur especially on stooping or a swelling may be noticed in the neck which diminishes after regurgitation causing dysphagia or cough

**Differential Diagnosis** A swelling in the neck which disappears after regurgitation of food is very suggestive An X ray examination after swallowing a barium paste usually confirms the diagnosis

**Course and Complications** A large diverticulum may cause œsophageal obstruction owing to pressure of its contents

**Treatment** Small diverticula are usually only diagnosed by X ray examination and require no treatment A larger diverticulum in the neck can be removed by operation The rarer varieties in the thorax are usually intractable Sometimes the patient can swallow more easily if he lies on his face

### Œsophageal Varices

Varicose veins in the lower part of the œsophagus are a common post mortem finding (see Fig 1) They are usually associated with cirrhosis of the liver, consequent on portal vein stagnation They may

also occur with obstruction of the inferior or superior vena cava, as in heart failure They are a cause of hæmatemesis or melæna and can be diagnosed during life by œsophagoscopy, or by a barium swallow, the film showing a characteristic appearance

### Œsophageal Ulceration

The following varieties of ulcer may occur Traumatic peptic simple as a complication of fevers such as diphtheria or scarlet fever, malignant, syphilitic, and tuberculous Peptic ulcers occur at the lower end of the œsophagus especially in association with thoracic stomach They cause pain on swallowing at the lower end of the sternum and in the back Vomiting and hæmatemesis may occur The diagnosis of ulceration is made by œsophagoscopy, and the treatment is as for gastric ulcer (see p 35) but olive oil m 60 to 120 should be given immediately before each feed

### Rupture of the Œsophagus

This may occur spontaneously when ulceration is present or from trauma caused by an ingested foreign body or the passage of an œsophagoscope or bougie It may rarely follow vomiting Thus in one of my cases a patient after rapidly drinking several glasses of beer vomited violently and was seized with acute pain in the left side of the chest and left flank He sat up and writhed in agony

*On Examination* The abdominal wall was rigid and a pleural rub was heard over the left lower chest in front The pulse was rapid and the temperature subnormal In a few hours signs of a left hydropneumothorax were present acid stomach contents were aspirated from the lower left chest and air was present above Death ensued in about 30 hours and at autopsy a rent was found in the œsophagus just above the diaphragm Cure has been reported by thoracotomy and repair of the tear

### Dilatation of the Œsophagus

This may be a uniform dilatation occurring above an œsophagea obstruction (see Dysphagia) or be due to achalasia of the cardia Localised dilatation occurs as a diverticulum

### Dysphagia

Definition Difficulty in swallowing

Etiology The causes may be classified as oral pharyngeal laryngea and œsophageal The most important causes are —

*Oral* Stomatitis ulcers of the tongue sore throat as in tonsillitis quinsy cleft palate palatal and pharyngeal paralysis as in diphtheria bulbar palsy myasthenia gravis and progressive muscular atrophy mumps fractured jaw arthritis of the jaw dislocated jaw

*Pharyngeal* Retropharyngeal abscess diverticulum syphilitic stenosis achalasia of the pharyngo œsophageal sphincter (Plummer Vinson syndrome)

*Laryngeal* Tuberculous laryngitis carcinoma of the larynx

**Course and Complications** In the majority of cases the illness is of short duration and complications apart from diarrhœa are rare In severe cases the sequelæ include ulceration of the stomach chronic gastritis and gastric stenosis

**Prognosis** This depends on the nature of the irritant Death is inevitable with phlegmonous gastritis and may occur rapidly in poisoning cases

**Treatment** If poisoning is suspected a specimen of the vomit should be saved in a clean jar for special tests to determine the nature of the irritant The patient should be put to bed and only sips of water allowed by mouth The bed should be warmed with hot bottles In cases of poisoning not due to a corrosive substance a stomach tube should be passed and the stomach washed out In poisoning due to corrosives the appropriate antidote should be given Subsequently only small quantities of water are allowed by mouth (see p 770)

In cases not due to poisoning a preliminary dose of castor oil fl oz ½ should be given followed by a gastric sedative such as Sod bicarb gr 10 bism carb gr 15, acid hydrocyan dil m 2 muc acac fl oz ½ aq ad fl oz 1 Ft oz 1 every 4 hours

To relieve epigastric pain, hot flannels or a mustard leaf may be applied If there has been much loss of fluid by vomiting rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose should be given every 4 to 6 hours In cases of severe collapse a stimulant such as Coramine 1 5 ml should be injected hypodermically and 4 fl oz of strong hot coffee may be administered per rectum

Food should be given with caution when vomiting and acute pain have ceased Three ounce feeds of citrated milk (gr 2 to fl oz 1) or arrowroot every 2 hours are most suitable

## Chronic Gastritis

**Etiology** It is doubtful whether many of the conditions which were formerly described as causing chronic gastritis do in reality have this effect Thus the stomach may be gastroscopically normal in many individuals who have consumed considerable amounts of alcohol for many years Chronic gastritis may be secondary to acute gastritis to local causes such as an ulcer or growth in the stomach or to passive venous congestion in association with cirrhosis of the liver and heart failure Chronic gastritis may also occur in wasting diseases such as carcinoma or tuberculosis or in blood diseases such as pernicious anæmia or leukæmia

**Pathology** Gastroscopic and other studies of the mucous membrane including gastric biopsy using a flexible tube reveal three types superficial atrophic and hypertrophic gastritis In superficial gastritis there is reddening œdema and exudation In atrophic gastritis the mucous membrane is thin and pale In hypertrophic gastritis the mucous membrane is velvety It may be nodular or polypoid and small erosions may be seen

**Clinical Findings** Superficial gastritis usually gives rise to no symptoms Atrophic gastritis may be met with in untreated pernicious

*Œsophageal* Internal Ingested foreign body such as a bone Intrinsic Œsophagitis ulcer stricture, spasm globus hystericus tumours, diverticula achalasia of the cardia paralysis (bilateral lesions of the vagus) External Enlarged cervical glands, tumours of the thyroid mediastinal tumours aneurysm of the aorta dilatation of the left auricle dissecting aneurysm congenital abnormalities such as right sided aortic arch double aortic arch and aberrant right subclavian artery pleural or pericardial effusion unilateral pulmonary fibrosis

## THE STOMACH

**Introductory** In the majority of cases of disorders of the stomach the physical signs are slight and inconclusive and diagnosis depends on a very careful investigation of the history and symptoms and on special investigations by test meals opaque meals and gastroscopy

### Gastritis

**Definition** Inflammation of the mucous membrane of the stomach This may be acute or chronic

### Acute Gastritis

**Etiology** Acute gastritis usually results from the ingestion of some irritant such as articles of food either indigestible, unmasticated or tainted (food poisoning) from excessive amounts of alcohol, from poisons such as perchloride of mercury or from the inhalation of an irritant gas In children it forms a part of summer diarrhœa (acute gastro enteritis) Acute gastritis may occur in influenza uræmia pneumonia, bronchitis, and rarely in typhoid or typhus fevers The infection here is probably hæmatogenous In pyæmia and small pox suppurative gastritis may occur

**Pathology** The mucous membrane of the stomach is red and inflamed and actual hæmorrhages may occur In infective cases there may be local or diffuse suppuration in the submucous tissues (phlegmonous gastritis) and this may cause perforation of the stomach wall

**Clinical Findings** The patient is usually an adult who gives a history of any of the causative conditions enumerated above He complains of being suddenly taken ill with pain in the epigastrium followed often by vomiting and thirst The vomit at first consists of stomach contents, but later there is usually little beyond mucus gastric juice and possibly blood In severe cases there are general toxic symptoms with prostration faintness pallor subnormal temperature and a rapid feeble pulse The temperature may be raised in cases due to food poisoning If the irritant enters the intestine there may also be diarrhœa

**Differential Diagnosis** The diagnosis is usually clear from the history and picture of the case The vomit should be examined in order to determine if possible the cause of the illness The acute abdominal pain may suggest abdominal angina or a tabetic crisis

### Gastric Flatulence and Aerophagy

*(Flatulent Dyspepsia)*

**Definition** Distention of the stomach with gas

**Etiology** Flatulence is frequently due to air swallowing (aerophagy), less often it is due to fermentative changes in the stomach

**Clinical Findings** The patient complains of epigastric distention after meals fulness and eructations of wind Flatulent dyspepsia is a prominent symptom in many cases of gall bladder disease It often gives rise to palpitations and pain near the apex of the heart which is mistaken by the patient for heart disease As a psychoneurosis aerophagy becomes a morbid habit, the patient constantly swallowing and belching up wind with noises which are very distressing for those near him Aerophagy may also occur with acute dilatation of the stomach

**Treatment** In flatulent dyspepsia the meals should be given dry and fluid drunk half an hour before or 2 hours after meals A charcoal biscuit may be eaten or a carminative such as ol cajaput m 2 or ol terebin m 10 may be taken on sugar or a mixture ordered containing Sod bicarb gr 10 sp ammon aromat m 20 sp chlorof m 5 infus caryophyll rec ad fl oz 1 Fl oz 1 three times daily after meals

In aerophagy due to nervous causes the nature of the air swallowing should be explained and the patient instructed to resist all desire to bring up' the wind Holding a cork between the teeth may prevent air swallowing

### Bulimia

A condition of excessive hunger which may be met with in diabetes mellitus gastric ulcer, hyperthyroidism gout or as a neurosis

### Anorexia Nervosa

A condition characterised by complete loss of appetite without any organic cause being discoverable The loss of appetite was attributed by Sir William Gull to a morbid mental state and it is thought by some that this leads to a functional disturbance of the anterior lobe of the pituitary The condition in some ways resembles Simmonds disease The latter however usually occurs at a later age period Predisposing causes include emotional crises unhappiness at home at school or at work slimming an operation or a severe illness The patient is usually a young woman aged 15 to 20 years who refuses to take any food steadily loses weight and suffers from amenorrhœa The amenorrhœa may begin before or at the same time as the anorexia There is an increased downy growth of hair on the trunk limbs lips and chin and the pubic hair may show the male distribution There is usually no loss of pubic or of axillary hair In severe cases there is œdema of the legs The patient is active and restless and displays complete indifference towards her symptoms Death may occur from what is apparently a functional disease

The nature of the illness should be explained to the patient and her relatives She should be isolated and small feeds given two hourly the

anæmia in iron deficiency anæmias and in gastric carcinoma The *symptoms are inconstant* but the patient may complain of dyspepsia and heartburn In hypertrophic gastritis the symptoms may resemble those of peptic ulcer and a severe hæmatemesis may occur

**Treatment** In atrophic gastritis the treatment appropriate for the anæmia should be given In hypertrophic gastritis the treatment is as for gastric ulcer

### The Gastric Dyspepsias

**Definition** Disturbances of stomach function

**Etiology** The main function of the stomach is to liquefy food and pass it on to the duodenum Disturbances of function may be secretory, muscular or nervous

### Hypochlorhydria and Achlorhydria

**Definition** Diminished secretion of gastric juice The term is usually employed to mean diminished secretion of hydrochloric acid

In achlorhydria there is absence of free hydrochloric acid and in achylia gastrica there is a complete absence of free hydrochloric acid and pepsin in the gastric juice

**Etiology** Hypochlorhydria may be met with in apparently healthy individuals Achlorhydria may also occur in apparently healthy individuals or may result from atrophic gastritis or carcinoma of the stomach It may also occur with simple achlorhydric anæmia Achlorhydria is more common in women It is rare with duodenal ulcer less uncommon with gastric ulcer Achylia gastrica is usually present with pernicious anæmia

**Clinical Findings** Frequently there are no symptoms In other cases the patient usually an adult complains of abdominal discomfort and fulness after a small meal flatulence anorexia heartburn and diarrhœa A test meal shows a complete absence of free hydrochloric acid and a low total acid curve The opaque meal shows rapid stomach emptying due to absence of acid in the duodenum Normally acid in the duodenum causes the pylorus to close until it is neutralised by the pancreatic secretion

**Treatment** This should be directed to the associated conditions such as anæmia It is doubtful whether hydrochloric acid given by mouth has any effect

### Hyperchlorhydria

**Definition** In hyperchlorhydria the volume of gastric juice may not be increased but the concentration of hydrochloric acid is greater than the normal of about *0 1 to 0 2% as obtained with the test meal*

**Etiology** Hyperchlorhydria occurs in about 5% of apparently normal men or in association with pylorospasm and juxta pyloric ulcer the gastric crises of tabes and at times in association with chronic cholecystitis or appendicitis

**Clinical Findings** No definite symptoms can be attributed to hyperchlorhydria

**Course and Complications** If untreated it may prove rapidly progressive

**Prognosis** This is serious and death may rapidly occur unless treatment proves successful

**Treatment** *Prophylactic* This consists in the avoidance of tight abdominal binders after operations Mouth washes should be used to prevent air swallowing

*Curative* If the condition is diagnosed sufficiently early and adequate treatment applied, it is not usually necessary to place the patient in the knee elbow position or on his face As soon as it is suspected a Ryle s tube should be passed through the nose and the stomach contents aspirated with a well fitting glass syringe If excessive fluid is obtained indicating dilatation the stomach should be kept empty by a continuous suction apparatus attached to the Ryle s tube Normal saline should be given intravenously by the continuous drip method to replace lost fluid and the patient should be allowed fluid by mouth as desired Physostigmine salicylate gr 1/200 or Prostigmin 1 mg should be given subcutaneously every 4 hours for 8 doses

## Chronic Dilatation of the Stomach

This may be non obstructive or obstructive

### Non obstructive Dilatation of the Stomach

It is doubtful whether the large stomach often associated with gastroptosis gives rise to a definite symptom complex

### Obstructive Dilatation of the Stomach

**Etiology** Obstructive dilatation may result from pyloric obstruction due to an ulcer new growth congenital stenosis, pylorospasm or external adhesions or to an hour glass stomach

**Pathology** The stomach is usually considerably enlarged and some hypertrophy of its musculature may occur

**Clinical Findings** The patient is often an adult male who complains of indigestion associated with gradual weakness There is epigastric discomfort flatulent distention and periodical vomiting of large quantities of sour fluid which has an offensive odour in malignant cases The vomit often contains articles of food eaten several days previously The appetite may remain good but constipation is severe

*On Examination* A marked stomach splash may be elicited several hours after the last meal and in some instances pyloric thickening is felt Visible peristalsis in which the waves pass from left to right in the upper abdomen may be noted after abdominal palpation and the outline of the distended stomach may be seen through a thin abdominal wall Occasionally antiperistaltic waves are seen A test meal shows stagnation of the stomach contents excess of mucus fermentation acids and a low or absent free hydrochloric acid content An opaque meal indicates the size of the stomach and delay in emptying This may be repeated after a course of tnc belladon m 15 t d s which is given for

nurse or doctor ensuring that each feed is eaten Feeding by *continuous* intragastric drip the tube being passed through the nose is very successful in some cases The patient cannot be considered to be cured until her monthly periods are restored

### Heartburn

(*Pyrosis*)

Heartburn is a burning sensation felt behind the sternum or in the epigastrium, usually accompanied by eructations or regurgitation of a little acid fluid into the mouth It often occurs in the early months of pregnancy, or in association with smoking or taking a meal when fatigued by the day s work In other instances it is associated with an organic disease of the œsophagus stomach or gall bladder Relief may usually be obtained by drinking a little warm water with half a tea spoonful of sodium bicarbonate dissolved in it

### Waterbrash

Waterbrash is a regurgitation of acid fluid into the œsophagus accompanied by a copious secretion of saliva The latter may run out of the mouth and be followed by vomiting It is usually associated with hypersecretion and relieved by alkalis

### Dilatation of the Stomach

(*Gastrectasis*)

*There are two types, acute and chronic*

### Acute Dilatation of the Stomach

**Etiology** The dilatation may be due to paralysis of the gastric branches of the vagus or to overactivity of the sympathetic associated with a kink of the third part of the duodenum where it is crossed by the superior mesenteric vessels This kink is produced by the drag of the dilated stomach Some writers believe that the dilatation is initiated by air swallowing It usually follows operations such as prostatectomy, but it may occur in any severe illness after childbirth after injuries to the spine femur or head or following a heavy meal

*Clinical Findings* Shortly after coming round from the anæsthetic after an abdominal operation the patient notices pain or oppression in the epigastrium and later large quantities of watery fluid containing mucus or bile pour out from the mouth

*On Examination* In the early stages there is slight fulness in both hypochondria Later the patient is pale and collapsed, the abdomen is distended and sometimes tender a stomach splash is elicited The pulse is rapid and of poor volume, and the temperature is usually subnormal

**Differential Diagnosis** Acute dilatation of the stomach must be differentiated from peritonitis or intestinal obstruction The absence of fever and fæcal vomiting together with the signs of a grossly dilated stomach are characteristic findings

tion The vomiting leads to alkalosis, with a raised plasma bicarbonate and a lowered plasma chloride figure

**Course and Complications** If untreated the baby usually dies in a few weeks from starvation recovery may take 1 to 3 months with medical treatment Gastro enteritis due to cross infection in a hospital ward is a serious complication

**Differential Diagnosis** The condition must be diagnosed from other causes of vomiting The characteristic features are the age of onset the projectile vomiting visible peristalsis and most important the palpable tumour In congenital duodenal stenosis the vomiting begins at birth and bile is present in the vomit

**Prognosis** A certain proportion of patients recover spontaneously but as it is impossible to say whether an individual one is going to do so treatment should be given immediately the diagnosis is made There is a difference of opinion as to whether this treatment should be medical or surgical and whether surgical treatment should only be given if medical fails On statistical evidence the palm must be awarded to the surgeons as in a series of 50 cases operated on in nursing homes 100% recovered and with a series of 100 cases who were *breast fed* before and after the operation and were operated on in hospital all recovered On the other hand with bottle-fed infants operated on in hospital a mortality of 10% must be expected death being usually due to gastro-enteritis acquired by cross infection in hospital

**Treatment** *Medical* This consisted formerly of gastric lavage using a No 6 or 7 Jacques soft rubber catheter and a 1% sodium chloride solution at 100° F, twice daily just before a feed Small hourly feeds were given of 1 to 2 teaspoonfuls of breast milk which had just been drawn off or of citrated or peptonised milk The feeds were gradually increased to 1½ oz The best medical results are obtained by the use of Eumydrin (atropine methylnitras) the mortality in Gothenburg being 1% whereas in England with hospital cases the mortality is about 11 5% between 70% and 80% being cured Eumydrin may be given by mouth using a 1 in 10 000 solution in water the first dose is 0 5 to 1 ml increasing by 0 5 ml at each feed until 2 to 3 ml are given six times daily The Eumydrin is administered half an hour before the feeds which are given three hourly The treatment has to be continued for about 4 weeks Eumydrin (0 6% in alcohol) may also be given by drops applied to the tongue One drop twice a day may prove sufficient to stop the vomiting It is a mistake to administer too much fluid by mouth or in the form of normal saline subcutaneously unless the patient is very dehydrated as this diminishes the effect of the drug The total fluid required (between feeds) is 3 oz per lb body weight during the first 24 hours subsequently the infant is given by mouth between feeds as much fluid as he requires in addition to his feeds Gastric lavage is not usually necessary Toxic effects include rise of temperature flushing of the skin and abdominal distension These can usually be relieved by omitting the next dose of Eumydrin The duration of the treatment is likely to be 28 days in hospital during which time there is a grave risk of gastro-enteritis occurring from cross infection unless

3 to 4 days to see whether the obstruction is due to pylorospasm Pylorospasm may be due to apprehension, and some radiologists believe that the relaxation of the spasm at the second *examination is due to the* relief of this apprehension rather than to the effect of the belladonna

**Course and Complications** Organic pyloric stenosis is progressive and if due to carcinoma usually rapidly so Tetany may occur as a complication

**Differential Diagnosis** Dilatation of the stomach can usually be distinguished from dilatation of the colon by clinical examination, and in *some cases of the latter removal of flatus by a rectal tube* establishes the diagnosis If the stomach is grossly dilated and filled with fluid ascites or an ovarian cyst may have to be eliminated in making the diagnosis There is usually no difficulty as the dilated stomach can be emptied by a tube and the swelling thus removed X rays also serve to establish the diagnosis

**Prognosis** This depends on the cause of the obstruction and its amenability to treatment

**Treatment** Gastro enterostomy is usually required in organic obstruction Stenosis due to spasm may be relieved by daily gastric lavage and the administration of tnc belladon m 15 t d s

### Congenital Hypertrophic Stenosis of the Pylorus

**Etiology** The stenosis is probably due to congenital hypertrophy of the circular fibres of the pylorus with superimposed pyloric spasm *Predisposing causes* 1 Age 2 to 4 weeks 2 Sex Males predominate in the proportion of four to one The first child of a family is especially prone to the affection Phimosis is probably of no etiological significance

**Pathology** *There is pyloric thickening with hypertrophy of the* circular muscle fibres the stomach may be dilated The duodenum is normal

**Clinical Findings** The patient is usually a baby boy either breast or bottle fed aged about 2 to 4 weeks who was healthy at birth but who has suffered from vomiting and constipation since he was about 2 weeks old More rarely the vomiting has been present from birth *The vomiting is forcible or projectile* the fluid being ejected 2 to 3 feet, and the baby loses weight The vomit does not contain bile

*On Examination* The baby is often pale, *a little cyanosed* and wasted and may appear dehydrated Visible peristalsis from left to right may be seen in the epigastrium after a feed A tumour can be felt in nearly every case if sufficient care is taken The warmed left hand is laid on the abdomen and the lower border of the liver defined Pressure is then made with the fingers in the region of the pylorus and after the baby is fed the small tumour due to the thickened pylorus can usually *be felt It may be under the liver* and felt only on inspiration and it may quickly relax An X ray photograph may be taken giving an ounce of milk and water and gr *60 of barium sulphate by a spoon* the delay in stomach emptying is thus revealed Gastric analysis usually

vomits up the blood Blindness temporary or permanent may follow hæmatemesis

*On Examination* The patient is often pale and blanched The vomited blood tends to be dark (coffee grounds) and is acid in reaction unless a large dose of alkali has been taken just before the vomiting Food may be present in the vomit and the blood is not aerated as in hæmoptysis The stools subsequently are dark and tarry (melæna) for about two days after the bleeding has ceased

**Treatment** This depends on the cause The routine treatment for hæmorrhage due to gastric ulceration is described on p 89

## Vomiting

**Definition** Expulsion of the stomach contents from the mouth by abdominal and diaphragmatic contractions

**Etiology** Vomiting may be due to —*Gastric Causes* Dyspepsia gastritis ulcer carcinoma congestion in heart failure or cirrhosis hour glass constriction pyloric obstruction emetics such as salt or mustard irritants such as arsenic digitalis poison gases etc

*Central Causes* Stimulation of the vomiting centre in the medulla by anæsthetics apomorphine tobacco toxins in uræmia alkalosis acidosis in diabetes mellitus and cyclical vomiting cholæmia preg nancy Graves disease Addison s disease scarlet fever, influenza acute yellow atrophy of the liver

*Nervous Causes* Emotions hysteria migraine concussion meningitis intracranial tumours abscess or hæmorrhage tabes dorsalis

*Reflex Causes* Pharyngeal irritation intestinal obstruction appendicitis worms peritonitis acute pancreatitis biliary colic renal colic Dietl s crisis vestibular causes as in Ménière s disease and possibly seasickness nasal causes due to odours uterine and ovarian irritation as in pregnancy testicular trauma

## Gastric Ulcer

(*Peptic Ulcer*)

**Definition** A simple ulcer of the stomach of doubtful etiology

**Etiology** The cause is unknown Gastric juice appears to be an essential factor in the ulcer production as peptic ulcer occurs only in situations exposed to its action such as the stomach first part of the duodenum last part of the œsophagus and in the jejunum after gastric anastomosis (anastomotic ulcer) Normally the gastric mucosa is protected from the corroding action of the acid by a thin layer of mucus Extensive skin burns may result in a Curling s ulcer This is an acute gastro duodenal ulcer situated near the pylorus A peptic ulcer may develop in an area of heterotropic gastric mucous membrane in a Meckel s diverticulum Other factors which may play a part in its production are Trauma from ingested irritant food Gastric arterial thrombosis or cardiac infarction Nervous disturbances such as over activity of the vagus or underactivity of the sympathetic which may result from tobacco Worry appears to predispose to hæmorrhage and

adequate precautions are taken as regards isolation and nursing If a favourable result is not obtained in a few days, an operation should be performed

*Surgical* The operation is that of Rammstedt s pyloromyotomy, the pylorus being divided longitudinally down to the mucous membrane It is usually performed under local anæsthesia the stomach being washed out immediately before the operation

### Hypertrophic Stenosis of the Pylorus in Adults

A few cases of hypertrophic stenosis of the pylorus of the congenital type have been met with in adults They are characterised by recur rent attacks of vomiting which may be associated with tetany without necessarily a history of infantile vomiting The condition can be cured by pyloroplasty, if feasible, or relieved by gastro enterostomy

### Cascade Stomach

This radiological abnormality is also known as the 'cup and spill' deformity or as the physiological hour glass stomach It probably results from an indentation of the posterior wall of the fundus of the stomach This may be due to distension of the splenic flexure with gas or to localised gastric spasm The barium fills the lower part of the fundal loculus and then spills down into the lower part of the stomach The patient may complain of recurrent attacks of epigastric pain nausea and vomiting No special treatment is required Volvulus of the stomach is probably a variant of the cascade stomach

### Hæmatemesis

**Definition** Vomiting of blood

**Etiology** The blood may be derived from various sites —

*The Stomach* (gastrorrhagia)

**Local causes** Erosion (gastrostaxis bleeding from minute foci) ulcer carcinoma or sarcoma hypertrophic gastritis trauma corrosives aspirin tablets swallowed whole simple tumours such as polypi or angiomata, a tuberculoma a gumma Intragastric rupture of aneurysm of the aorta Tabes dorsalis (gastric crises)

Portal congestion due to cirrhosis hepatis splenic anæmia heart failure thrombosis of the portal vein

Toxic and infective causes Yellow fever Weil s disease small pox hæmorrhagic diphtheria hæmorrhagic scarlet fever hæmor rhagic measles appendicitis cholecystitis septicæmia influenza cholæmia uræmia

Blood diseases Leukæmia purpura pernicious anæmia hæmo philia erythæmia

*The Œsophagus* Ruptured varicose veins in cirrhosis of the liver

*The Duodenum* An ulcer

*The Lungs Nose or Mouth* The blood from these sources is swallowed before it is vomited

**Clinical Findings** If the hæmatemesis is severe the patient usually experiences a preliminary feeling of faintness with nausea, and then

be found in the epigastrium the tender spot corresponding with the site of the pain as demonstrated by the patient with his finger tip Some muscular rigidity of the upper rectus muscle may be felt on one or both sides An opaque meal may demonstrate the ulcer as a projecting mass of barium in the lesser curvature in other cases a niche or permanent filling defect may be seen in the stomach wall Pyloric spasm may be present with juxta pyloric ulceration An incisura or hour glass constriction may be noted in some cases with mid gastric ulcers If a constriction of the stomach wall is noticed the opaque meal should be repeated after the patient has been given a course of bella donna such as tnc belladon m 15 t d s a c for 3 to 4 days to see if the constriction is due to spasm or to organic deformity A muscular spasm is often seen in the stomach wall opposite the site of the ulcer A fractional test meal shows a climbing type of curve with high acidity if the ulcer is near the pylorus and pylorospasm is present Blood may be found in the specimens removed In at least 50% of cases the test meal findings are normal especially if the ulcer is not juxta pyloric In some cases gastroscopy will reveal an ulcer which is not demonstrable radiologically the converse is also true A gastro jejunal stoma is not always easy to see The fæces The occult blood test may be positive Using the modified Gregersen benzidine test there is no necessity to exclude meat with the exception of liver and black pudding or green vegetables from the diet There are many atypical cases in which the history and symptoms do not suggest gastric ulcer and yet its presence is revealed by the opaque meal

**Differential Diagnosis** In very many cases a presumptive diagnosis of gastric ulcer can only be made after X ray and test meal examinations the symptoms and signs being entirely misleading In other cases the diagnosis is established by gastroscopy or laparotomy Such conditions as gastritis carcinoma of the stomach tabetic crises chronic appen dicitis chronic cholecystitis and gastric neuroses may all cause symptoms closely resembling those of gastric ulcer Carcinoma should be suspected if the pain is not relieved by a week s strict treatment

**Course and Complications** *Acute gastric ulcers* These usually heal rapidly when the appropriate treatment is carried out but a second ulcer has been known to form during the first stage of treatment *Chronic gastric ulcers* These pursue an intermittent course They may heal spontaneously or progress and cause such complications as hæmorrhage perforation localised or general peritonitis gastric stenosis due to pyloric or hour glass constriction perigastric abscess or adhesions or carcinoma General complications are anæmia and malnutrition

**Prognosis** This is on the whole good A gastric ulcer rarely proves fatal apart from such complications as perforation hæmorrhage or malignant change Efficient medical treatment is improving the ultimate prognosis

**Treatment** In the majority of cases the patient requires a preliminary 4 to 6 weeks course of treatment in bed followed by a prolonged after treatment The teeth should be attended to after dental radiograms have been taken any focus of sepsis being eradicated

perforation Stimulation of a vegetative centre in the inter brain by Pituitrin or by a tumour with consequent increased vagal activity, may be a factor The posterior lobe of the pituitary in animals produces a gastrotoxic substance which can cause lesions in the acid bearing area of the stomach There may also be a gastric ulcer diathesis as gastric ulcers tend to run in some families and duodenal ulcers in others

**Pathology** The ulcer varies in size from about 4 to 80 mm in diameter, but it may be larger, it is usually about the size of a shilling (24 mm in diameter) and is commonly circular or oval Multiple ulcers may occur Acute and chronic ulcers are described The former are usually small and often multiple, their floor is smooth and adhesions *are not found They may form rapidly in four or five days* In chronic ulcers there may be much fibrosis producing pyloric stenosis or hour glass constriction of the stomach The following are the most *frequent sites Near the pylorus, on the lesser curvature on the* posterior wall on the anterior wall and cardia on the greater curvature The ulcer is sharply delineated and extends a variable distance through the stomach wall *It may erode and perforate the stomach wall, or be* surrounded by inflammatory tissue or become adherent to such organs as the pancreas

Other changes found in association with gastric ulcer are perforation of an artery in the ulcer, perigastric suppuration due to localised peritonitis which may cause a subphrenic abscess generalised peritonitis from perforation, fistulæ with the colon duodenum or pleura and peri gastric adhesions to the liver gall bladder, etc Carcinoma rarely develops in an old gastric ulcer, but healing with a resultant scar is not infrequent

**Clinical Findings** The patient who has an acute ulcer is *often an* anæmic girl the first symptom being hæmatemesis Chronic ulcers are equally common in males or females The patient is usually an *adult over the age of 20 although a gastric ulcer has been recorded in a* child aged three months There is a history of periodic attacks of indigestion The characteristic features of the attack are epigastric *pain appearing* a quarter of an hour to one hour after food and disappear ing before the next meal The pain is believed to be caused by muscular contractions or spasm of the stomach It may be felt in the epigastrium to the left of the mid line or near the xiphisternum and may pass through to the back near the angle of the left scapula It is often relieved by vomiting or by alkali medicines Other symptoms such as nausea, vomiting, hæmatemesis or melæna may occur In some instances, especially when the ulcer is on *the lesser curvature of the* stomach or near the cardiac end there are no symptoms until a severe hæmatemesis occurs When the ulcer is situated close to the pylorus, *the symptoms resemble those of a duodenal ulcer* The appetite is often good but the patient is afraid to satisfy it Other symptoms such as heartburn or flatulence with epigastric distention may be troublesome

*On Examination* The patient is often well nourished but the tongue is usually furred Tenderness and cutaneous hyperalgesia may

be found in the epigastrium the tender spot corresponding with the site of the pain as demonstrated by the patient with his finger tip Some muscular rigidity of the upper rectus muscle may be felt on one or both sides An opaque meal may demonstrate the ulcer as a projecting mass of barium in the lesser curvature in other cases a niche or permanent filling defect may be seen in the stomach wall Pyloric spasm may be present with juxta pyloric ulceration An incisura or hour glass constriction may be noted in some cases with mid gastric ulcers If a constriction of the stomach wall is noticed the opaque meal should be repeated after the patient has been given a course of bella donna such as tnc belladon m 15 t d s a c for 3 to 4 days to see if the constriction is due to spasm or to organic deformity A muscular spasm is often seen in the stomach wall opposite the site of the ulcer A fractional test meal shows a climbing type of curve with high acidity if the ulcer is near the pylorus and pylorospasm is present Blood may be found in the specimens removed In at least 50% of cases the test meal findings are normal especially if the ulcer is not juxta pyloric In some cases gastroscopy will reveal an ulcer which is not demonstrable radiologically the converse is also true A gastro jejunal stoma is not always easy to see The fæces The occult blood test may be positive Using the modified Gregersen benzidine test there is no necessity to exclude meat with the exception of liver and black pudding or green vegetables from the diet There are many atypical cases in which the history and symptoms do not suggest gastric ulcer and yet its presence is revealed by the opaque meal

**Differential Diagnosis** In very many cases a presumptive diagnosis of gastric ulcer can only be made after X ray and test meal examinations, the symptoms and signs being entirely misleading In other cases the diagnosis is established by gastroscopy or laparotomy Such conditions as gastritis carcinoma of the stomach tabetic crises chronic appen dicitis chronic cholecystitis and gastric neuroses may all cause symptoms closely resembling those of gastric ulcer Carcinoma should be suspected if the pain is not relieved by a week s strict treatment

**Course and Complications** *Acute gastric ulcers* These usually heal rapidly when the appropriate treatment is carried out but a second ulcer has been known to form during the first stage of treatment *Chronic gastric ulcers* These pursue an intermittent course They may heal spontaneously or progress and cause such complications as hæmorrhage, perforation localised or general peritonitis gastric stenosis due to pyloric or hour glass constriction perigastric abscess or adhesions, or carcinoma General complications are anæmia and malnutrition

**Prognosis** This is on the whole good A gastric ulcer rarely proves fatal apart from such complications as perforation hæmorrhage or malignant change Efficient medical treatment is improving the ultimate prognosis

**Treatment** In the majority of cases the patient requires a preliminary 4 to 6 weeks course of treatment in bed followed by a prolonged after treatment The teeth should be attended to after dental radiograms have been taken any focus of sepsis being eradicated

A modified Sippy diet is advisable with a neutralising substance after feeds, and olive oil, belladonna or atropine before the feeds as described later The treatment recommended is as follows —

Healing Stage (Diet 1) *Week* 1 Feeds of 5–8 oz are given at 7 a m, 9 a m 11 a m 3 p m, 5 p m 7 p m, 9 p m, 11 p m and once during the night, if awake The feeds consist of milk (warm and containing gr 3 sodium citrate to the ounce) Horlick s malted milk or Benger s food At least 5 of the feeds should be of milk which may be coloured with tea Sugar may be added to the feeds, as desired A raw egg should be added to one or more of the milk feeds Strained orange juice, tomato juice or blackcurrant purée 1 oz t i d or ascorbic acid tablets, mg 50 t i d should be given daily throughout the treatment to supply vitamin C If the patient is hungry ½ oz of glucose may be taken t i d

*Week* 2 A little thin bread and butter or a slice of dry toast with butter is given with two feeds, and a rusk or a plain biscuit may be taken with the other feeds In addition the patient may be given twice a day one of the following Sweet jelly milk jelly custard or junket and once a day mashed potatoes or sieved vegetables The feeds are still given two hourly

*Week* 3 The feeds are given every 2½ hours from 7 a m to 10 p m They consist of 5–8 oz milk every other feed with 1 egg in one or more of the feeds, the thin bread and butter toast, rusk or plain biscuit and sugar as in Week 2 The alternate feeds are composed of a similar quantity of potato or tomato soup, arrowroot cornflour or milk pudding (sago or tapioca) and once a day a little milk jelly sweet jelly custard or junket apple or prune purée cream or honey Further additions to the diet are a small helping of porridge made from very fine oatmeal, pounded fish a lightly boiled egg sponge or Madeira cake

*Weeks* 4 *to* 6 Feeds are still given every 2½ hours Add a choice of minced meat scrambled egg sweetbread brains or tripe daily Ice cream or Yoghurt may also be taken

Immediately before 3 feeds ½ fl oz of olive oil is given and directly before 3 other feeds Tnc belladon m 5 to 10, aq chlorof ad fl oz ½ or Atropin sulph *gr* 1/200 *in m* 60 water is given The neutralising substance used may be magnesium trisilicate a teaspoonful after feeds or a 10% aluminium hydroxide gel, Aludrox m 60 in water, 6 times a day 1 hour after feeds and 2 teaspoonfuls the last thing at night or Gelusil 2 tablets after feeds and last thing at night During the third and fourth weeks the neutralising substance is taken 3 times a day after feeds, and a double dose at night In the fourth to sixth weeks the neutralising substance is taken twice a day after feeds and a double dose at night The patient should have throughout the treatment a milk feed by his bed to drink at night if he wakes The mouth should be cleansed after each feed with sod bicarb gr 60 in 5 fl oz of water to prevent parotid suppuration No smoking is allowed

A repeat X ray examination should be made at the end of 6 weeks If the ulcer has not healed the above diet should be continued for

**Convalescent Stage (Diet 2)** During this period of about a month the patient is allowed up for gradually increasing periods and additions are made to the dietary

7 30 *a m* *Breakfast* One egg (lightly boiled) or steamed fish thin bread and butter or crisp toast and butter (to be well chewed) honey or apple jelly sugar and milky tea

10 *a m* Milk 5 oz or Horlick's malted milk or Benger s food

12 30 *p m* *Lunch* Milk soup (potato or artichoke) fresh white fish (boiled or steamed) or rabbit chicken or tender mutton mashed potato Custard or milk pudding steamed or baked pudding trifle meringue soufflés honeycomb mould or stewed apples (with no pips skin or core) or prune purée and the juice of an orange

3 *p m* Thin bread and butter or biscuits and butter, and one small slice of sponge or Madeira cake and milky tea

5 30 *p m* Milk 5 oz or Horlick s malted milk

8 *p m* *Dinner* As for lunch

10 30 *p m* Milk 5 oz (containing gr 15 sodium citrate) or Benger s food

The neutralising agent should be taken after breakfast lunch and dinner and last thing at night and the olive oil or belladonna before breakfast lunch and dinner The meals should be small and eaten slowly After about a month the after treatment is commenced

**After treatment (Diet 3)** 8 *a m* *Breakfast* A selection from porridge made from very fine oatmeal egg boiled poached or scrambled white fish grilled or steamed cold ham or tongue toast crisply made and well chewed breakfast biscuits honey apple jelly butter sugar cream and milky tea or coffee

11 *a m* A glass of milk Ovaltine Horlick s malted milk or Benger s food and a biscuit

1 *p m* *Lunch* A selection from soup made from milk and vegetables such as potato or artichoke Fresh white fish boiled grilled or steamed with white sauce or fish soufflé Meat such as minced fresh beef mutton or veal grilled lamb cutlet boiled or roast chicken sweetbreads boiled tripe or boiled ham Vegetables such as mashed potato steamed or boiled green vegetables passed through a sieve and served with butter marrow peas if tender Sweets such as custard boiled or baked junket milk pudding very well cooked cornflour stewed apples with no pips core or skin chocolate soufflé plain steamed or baked pudding Crisp toast the juice of one or more oranges, cheese soufflé

4 *p m* A cup of milky tea thin bread and butter spongecake or plain cake honey or jelly jam

7 *p m* *Dinner* As at lunch

10 *p m* A feed as at 11 a m

The breakfast lunch and dinner should be small meals well chewed and eaten slowly The patient should rest for half an hour after meals No condiments or vinegar should be eaten and nothing taken which cannot be reduced to a soft pulp in the mouth If it is impossible to obtain the liquid feeds between all the meals a biscuit or some chocolate

should be eaten A dose of the neutralising agent should be taken every night on retiring to bed

*The following articles of food are forbidden* Smoked salmon kippers whitebait tough meat pork, high game sausages brawn, curry made up dishes, fried foods cheese (except cream cheese) meat extracts and meat soups, pickles, salads, uncooked vegetables, celery carrots cucumber onions or any fibrous vegetables new or wholemeal bread buns, Ryvita Vitawheat digestive and oatmeal biscuits hot buttered toast unripe or raw fruit rhubarb plums, nuts, raisins, sultanas figs jam with pips marmalade with peel, strong tea or coffee aerated drinks and alcohol

Cases of gastric ulcer which do not respond to strict treatment with the modified Sippy diet, should be tried with a continuous intragastric drip A Ryle s tube is passed through the nose into the stomach and joined by a glass connection to rubber tubing attached to the drip The fluid flows at a rate of 40 drops a minute day and night, *i e* 6 pints in 24 hours The fluid used consists of milk 1 pint, dextrose 2 oz and sod citrate gr 10 To this, Aludrox ½ fl oz should be added if test samples removed from the stomach by day or night, contain free HCl The Ryle s tube is changed every other day cleaned and boiled, and the rubber tubing and glass connections are also washed and boiled This treatment may be continued for 3 to 6 weeks

**The Medical Treatment of Perforation** Some cases of perforation have been treated successfully without operation The treatment is applicable in early cases before the peritoneal cavity is flooded Morphine gr ¼ is injected intravenously, repeated if necessary and an Amethocaine lozenge gr 1 is sucked The stomach is emptied with a Senoran s evacuator and a large tube A small stomach tube is now passed through the nose into the stomach and left there the stomach being emptied by aspiration with a 20 ml syringe every half hour The fluid balance is maintained by rectal subcutaneous or intravenous injection of normal saline During the second 24 hours aspirations are made hourly followed by drinks of 1 oz of water On the third day milk and water are given by mouth and the tube removed when the fluid output equals the intake showing that the fluid is not collecting in the stomach Subsequent treatment is as for acute peptic ulcer

**The Indications for Surgical Treatment** These are as follows Perforation Organic pyloric stenosis or hour glass constriction which impedes stomach emptying Perigastric abscess Intractable hæmorrhage due to a perforated sclerosed artery which will not repair by coagulation A suspicion of carcinoma as aroused by the severity of the pain loss of weight the persistence of blood in the fæces and the progressive fall in the free hydrochloric acid in the fractional test meal Repeated hæmorrhages not responding to medical treatment or recurrent gastric ulcers may also require operation Partial gastrectomy is usually the operation of choice for gastric ulcer

**The Medical Treatment of Severe Hæmatemesis** The patient should be put to bed and providing the hæmatemesis is not due to [illegible] of the liver a subcutaneous injection of morphin sulph

gr ¼ and atropin sulph gr 1/100 given This may be repeated up to a total of gr 1 morphine in the 24 hours In very severe cases nothing must be swallowed by mouth until the bleeding has stopped as judged by the absence of hæmatemesis or melæna The mouth may be washed out with a little cold water An hourly pulse chart should be kept The blood pressure and hæmoglobin percentage should be determined every 12 to 24 hours The blood urea should be estimated and if raised determinations should be made daily until it falls to normal The bowels should not be opened for 4 to 7 days an enema then being given Rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose should be given every 4 to 6 hours or a rectal drip may be used with a Murphy vulcanite rectal nozzle which allows free escape of flatus Six to 8 pints of normal saline (0 85% sodium chloride) and isotonic dextrose (5%) should be run in slowly every 24 hours resting the bowel every other hour by interrupting the flow Blood transfusion is required if the hæmoglobin is below 40% the systolic blood pressure below 90 mm Hg the pulse rate over 140 or the blood urea over 100 mg per 100 ml One pint of blood will increase the hæmoglobin by about 8 to 10% and it should be raised to about 80% By the drip method a 10% increase in hæmoglobin should be achieved every 4 hours If there is a history of previous severe hæmorrhages or if there is arteriosclerosis and the bleeding persists an operation will probably be required This should not be delayed until the patient is exsanguine and a preliminary blood transfusion should be given before the operation After 2 to 4 days and when the bleeding has been arrested as judged by absence of hæmatemesis and by the general condition of the patient 4 fl oz of half strength normal saline (0 42% sodium chloride) should be given by mouth every 4 hours for 24 hours Milk feeds are now begun first milk 1 oz water 1 oz and emuls mag oxid m 30 every 2 hours and then milk 2 oz every 2 hours gradually increased to 5 oz every 2 hours Tnc belladon m 10 to 15 is given before 3 feeds and olive oil fl oz ½ alternately before 3 other feeds A teaspoonful of the neutralising powder is given after 5 feeds and a double dose at night Ferri et ammon cit gr 30 should be given t i d between feeds The patient should now begin the routine treatment for gastric ulcer A barium meal should not be given within 2 months of a hæmatemesis as it may provoke a recurrence

**Routine Medical Treatment of Hæmatemesis** In the majority of cases of hæmatemesis the patient does better with early feeding than with starvation The true Meulengracht diet is unsuitable for patients in this country but Witts diet usually proves very efficacious The diet is shown on p 40

The milk is not citrated but may be flavoured with Horlick s malted milk or Ovaltine Vitamin C is given as fruit juice or as ascorbic acid tab mg 50 t i d Sips of water or of dextrose solution or half strength normal saline are allowed between feeds up to 5 oz an hour The total fluid intake in the 24 hours should be 5 pints Neutralising substances are only given if there is abdominal pain No purgatives are

administered, and if the bowels are not opened an enema is given on the seventh day Transfusion is given according to the criteria laid down above (see p 39)

| Feeds by day | Food | | D y | | |
|---|---|---|---|---|---|
| | | | 1 | | 3 and subsequent. |
| 1 | Whole milk (fresh or dried) | oz | 5 | 5 | 5 |
| | Patent barley or strained porridge | | Portion | Portion | Portion |
| 2 | 1 egg beaten up in milk | oz | 5 | 5 | 5 |
| | Buttered rusks or cream crackers | | – | 1 | 2 |
| 3 | Whole milk (fresh or dried) | oz | 5 | 5 | 5 |
| | Marmite to taste | | – | – | – |
| | Barley sugar | oz | 1 | 1 | 1 |
| | Thin crustless white bread and butter slices | | – | 1 | 2 |
| 4 | Strained orange or tomato juice | oz | 1 | 1 | 1 |
| | Vegetable purée | | Portion | Portion | Portion |
| | Pudding | | | | |
| | Cream | oz | 1 | 1 | 1 |
| | Boiled or steamed fish | | – | – | Portion |
| 5 | 1 egg beaten up in milk | oz | 5 | 5 | 5 |
| | Barley sugar | oz | 1 | 1 | 1 |
| | Buttered rusks or cream crackers | | – | 1 | 2 |
| 6 | Whole milk (fresh or dried) | oz | 5 | 5 | 5 |
| | Fruit purée | | Portion | Portion | Portion |
| | Pudding | | | | |
| | Cream | oz | – | 1 | 1 |
| | Thin crustless white bread and butter slices | | – | 1 | 2 |
| 7 | 1 egg beaten up in milk | oz | 5 | 5 | 5 |
| | Black treacle or barley sugar | oz | 1 | 1 | 1 |
| | Buttered rusks or cream crackers | | – | 1 | 2 |
| 8 | Whole milk (fresh or dried) | oz | 5 | 5 | 5 |
| | Fruit purée | | Portion | Portion | Portion |
| | Pudding | | | | |
| Feeds at night (when awake) | 1 Whole milk (fresh or dried) | oz | 5 | 5 | 5 |
| | 2 1 egg beaten up in milk | oz | 5 | 5 | 5 |
| Between feeds | Strained orange or tomato juice | oz | 1 | 1 | 1 |
| | Approximate calorific value | | 2 545 | 3 118 | 3 624 |

## Gastric Diverticula

A diverticulum of the stomach is relatively uncommon It is usually a congenital abnormality and in the majority of cases is situated near the cardia on the lesser curvature or posterior wall of the stomach

**Clinical Findings** The patient may complain of epigastric discomfort or pain and of recurrent attacks of severe vomiting Hæmatemesis is rare The diagnosis is made by X ray examination

**Treatment** If the symptoms are severe the diverticulum can be resected or invaginated

## Carcinoma of the Stomach

**Etiology** The cause is unknown Most recent statistics tend to show that carcinoma rarely develops from a chronic gastric ulcer

Chronic atrophic gastritis is an important predisposing condition Carcinoma ventriculi is two to three times as common in the male as in the female sex It is estimated that 4% of all adults die from it

Pathology The following types are described 1 Scirrhous a spheroidal celled carcinoma with much fibrous stroma 2 Medullary or encephaloid, a spheroidal celled carcinoma with little fibrous stroma 3 Adeno carcinoma, a columnar celled growth Dual (kiss) adeno carcinoma has been described occurring on apposing surfaces of the stomach In the so called Krukenberg tumour secondary deposits occur in the ovary 4 Leather bottle stomach or *linitis plastica* a diffuse scirrhous growth 5 Colloid the growth tends to spread to the omentum 6 Malignant degeneration of a peptic ulcer 7 Squamous celled carcinoma is rarely seen and then it is at the cardiac end of the stomach

The growth is most often situated at the pylorus then on the lesser curvature near the cardia on the posterior wall or diffusely on the anterior wall Adeno carcinoma may occur as a stomal growth following a gastro enterostomy Secondary carcinoma of the stomach is rare the primary lesion usually being in the breast

Clinical Findings The patient is often an adult male over the age of 40 He states that he has recently suffered from indigestion a complaint formerly unknown to him or not noticed for several years The earliest symptoms are usually abdominal discomfort or pain not definitely related to food and often present at night, followed by anorexia nausea and later pain aggravated by food heartburn eructations flatulence vomiting which may be very persistent regurgitation of mucus general weakness anæmia constipation, loss of weight and difficulty in digesting solid food In carcinoma of the cardia dysphagia is often the presenting symptom In other cases the early symptoms suggest a duodenal ulcer but without the typical periods of remission Recurrent thromboses of veins in the legs may be an early symptom Hæmatemesis is a rare occurrence but when present the vomit may show the typical coffee grounds appearance I have however seen cases in which recurrent hæmatemesis was the only localising symptom The pain later becomes very severe

*On Examination* In early cases nothing abnormal can be found beyond epigastric tenderness As the disease progresses cachexia becomes noticeable, a palpable tumour due to the growth may be felt in the epigastrium or left hypochondrium and enlarged glands may be felt above the left clavicle (Virchow s gland due to spread along the thoracic duct) in the left axilla and in the groins Nodules may be felt in the abdominal wall around the umbilicus The liver may be enlarged and irregular due to secondary deposits and ascites may result from pressure of enlarged glands in the portal fissure Rectal examination may reveal secondary deposits on the shelf of Blumer In any case in which the suspicion of gastric carcinoma arises the following investigations should be carried out —Opaque meal This may show a large filling defect due to the growth protruding into the stomach and giving rise to gastric stenosis or in early cases it is due to an interference

with the normal peristaltic wave at a certain spot in the stomach as seen on the screen In all cases the patient should be X rayed in the Trendelenberg position to show up the fundus of the stomach Fractional test meal The typical findings are foul dark resting stomach contents with some blood and high fermentation acid but no free hydrochloric acid Evidence of stagnation is shown by the presence of charcoal particles from the milk and charcoal feed given the night before Subsequent specimens show achlorhydria (absence of free hydrochloric acid) which is at times histamine fast and the fermentation acids remain on the high side Free hydrochloric acid may be found in 30% of cases Achlorhydria is probably due to previous gastritis and not to the growth involving the acid secreting area The lactic acid in the stomach contents results from fermentation and is not a specific product of the growth Malignant cells may be found in the stomach washings In some cases in which the X ray diagnosis of carcinoma is doubtful, a definite diagnosis either positive or negative can be made by gastroscopy Occult blood in fæces A specimen of the fæces should be tested for occult blood (see p 35) The blood There is usually severe anæmia which may approximate to the pernicious type, with some megalocytes and an occasional megaloblast, but the colour index is not usually above unity There is usually a leucocytosis of 12 000 per c mm or over Secondary deposits in bone marrow may cause a leuco erythroblastic anæmia

**Differential Diagnosis** The most important conditions to exclude are Dyspepsia from other causes, including ulcers of the stomach and duodenum Authors of text books are often blamed for only giving a picture of advanced and inoperable carcinoma of the stomach It should be suspected in all cases of digestive disturbances which do not quickly yield to adequate treatment In all cases of doubt, full investigations as described above should be carried out Even then in some cases the diagnosis remains uncertain and can only be established by biopsy Carcinoma of the stomach does at times occur in young adults so it cannot be excluded on the ground of age alone Pernicious anæmia A blood and bone marrow examination should exclude pernicious anæmia in which an achylia gastrica (absence of hydrochloric acid and pepsin) is almost always present Splenic anæmia A swelling in the left hypochrondrium associated with a hæmatemesis and anæmia may suggest an enlarged spleen and splenic anæmia The opaque meal usually serves to differentiate A gumma of the stomach The Wassermann reaction will help to exclude this, and if positive the response to vigorous anti syphilitic treatment as described on p 596 affords further confirmatory evidence

**Course and Complications** Death usually occurs within 6 to 12 months from the date of diagnosis unless early surgical intervention proves successful Complications include dilatation or rupture of the stomach gastro colic fistula jaundice due to obstruction of the bile or hepatic ducts by secondary deposits pleural effusion and thrombosis of the femoral vein

**Prognosis** This is usually hopeless unless early operation has been

performed Malignancy is greatest in poorly differentiated tumours Polypoid tumours grow more slowly than do infiltrating ones

Treatment If the investigations point to early carcinoma laparotomy should be performed and the growth removed surgically if possible or radium may be applied locally Operation in the presence of clinical evidence of metastases such as enlarged glands or liver is not justifiable except to relieve pyloric obstruction Deep X ray treatment does not effect a cure

Medical treatment consists in —Diet The patient should be given what he fancies If there is gastric obstruction liquids or semi solids only should be allowed Gastric lavage In cases of pyloric obstruction which are not operated on the stomach should be washed out with sodium bicarbonate solution (gr 60 in fl oz 20) daily or more frequently using a rubber tube Drugs These are required for pain Nepenthe m 20 to 30 t i d may be given by mouth combined with aspirin gr 10 to 15 or Largactil mg 25 tab t i d Pethidine hydrochlor mg 100 Physeptone mg 10 or Heptalgin mg 10 may be injected intramuscularly two or three times a day These drugs can also be given in tablet form by mouth but are not then so efficacious The tablets contain pethidine hydrochlor mg 50 Physeptone mg 5 and Heptalgin mg 10 To secure sleep hypodermic injections of morphin sulph gr ½ to 1 or Omnopon gr 1/3 are usually required The anæmia usually improves temporarily with liver treatment (as for pernicious anæmia see p 516)

### Sarcoma of the Stomach

It is stated that about 1% of malignant growths in the stomach are sarcomatous Growth is rapid The age incidence is usually between 30 and 40 Clinically the condition can only be distinguished from carcinoma by operation Secondary metastatic sarcoma may occur in the gastric wall

### Benign Tumours Cysts, and Foreign Bodies in the Stomach

These include Polypus (adenomatous tumour) fibroma myoma adenomatous cyst hydatid cyst and hair balls (trichobezoars) In gastric polyposis the tumours are composed of hypertrophied mucous membrane The swellings are globular and arranged in rows in the transverse axis of the stomach The symptoms are usually due to bleeding ulceration or obstruction There may be a history of dyspepsia for several years Intermittent pyloric obstruction occurs in about 10% of cases The barium meal shows a typical mottled appearance those areas not showing the shadow of barium being occupied by the polypi The barium meal should always be repeated after washing out the stomach to make sure that the appearances are not caused by food residue

The treatment of these tumours is surgical

### Tuberculosis of the Stomach

The stomach is very rarely affected by tuberculosis An ulcer may occur secondary to pulmonary tuberculosis or miliary tubercles may be found in the mucous membrane

with the normal peristaltic wave at a certain spot in the stomach, as seen on the screen In all cases the patient should be X rayed in the Trendelenberg position to show up the fundus of the stomach Fractional test meal The typical findings are foul dark resting stomach contents with some blood and high fermentation acid, but no free hydrochloric acid Evidence of stagnation is shown by the presence of charcoal particles from the milk and charcoal feed given the night before Subsequent specimens show achlorhydria (absence of free hydrochloric acid) which is at times histamine fast and the fermentation acids remain on the high side Free hydrochloric acid may be found in 30% of cases Achlorhydria is probably due to previous gastritis and not to the growth involving the acid secreting area The lactic acid in the stomach contents results from fermentation and is not a specific product of the growth Malignant cells may be found in the stomach washings In some cases in which the X ray diagnosis of carcinoma is doubtful a definite diagnosis either positive or negative can be made by gastroscopy Occult blood in fæces A specimen of the fæces should be tested for occult blood (see p 35) The blood There is usually severe anæmia which may approximate to the pernicious type with some megalocytes and an occasional megaloblast but the colour index is not usually above unity There is usually a leucocytosis of 12 000 per c mm or over Secondary deposits in bone marrow may cause a leuco erythroblastic anæmia

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**Prognosis** This is usually hopeless unless early operation has been

is a high climbing acid curve, due to pyloric spasm and rapid emptying due to violent gastric contractions If there is pyloric stenosis the high acidity persists with delay in stomach emptying The occult blood test The fæces usually give a positive occult blood test, if active ulceration is present

**Differential Diagnosis** Many cases of duodenal ulcer are atypical A gastric and duodenal ulcer may coexist as demonstrated by X rays The symptoms may be those of gastric ulcer whereas the X ray examination indicates an ulcer in the duodenum The duodenal symptoms may be due to duodenitis or reflex associated with chronic cholecystitis appendicitis or renal lesions The symptoms may be typical of cholecystitis although the gall bladder is normal and a duodenal ulcer is present Duodenal adhesions may cause an X ray deformity of the duodenal cap

**Course and Complications** There are various possibilities when a duodenal ulcer exists They are —Complete recovery without further symptoms or in healing pyloric stenosis may develop with progressive vomiting Progressive ulceration, with adhesions Perforation This may be a minute leak with very localised peritonitis, or a subphrenic abscess or generalised peritonitis may ensue Hæmorrhage Chronic pancreatitis with a rise of urinary diastase up to about 100 units, is met with in some cases Duodenal ulcers do not become malignant

**Prognosis** This depends upon early recognition and adequate treatment There is however a tendency to recurrence

**Treatment** The medical treatment is identical with that detailed for gastric ulcer (see p 35) *Surgical Treatment* Gastro enterostomy is necessary for organic stenosis Gastro enterostomy alone is contra indicated when the gastric acidity is high owing to the risk of subsequent development of anastomotic ulcer In such cases a gastrectomy of the acid bearing portion of the stomach should also be performed An alternative operation is vagotomy combined with pylorectomy

## The Dumping Syndrome

This name is applied to a group of symptoms which may occur after partial gastrectomy or gastro enterostomy The more extensive the resection of the stomach the more likely is dumping to occur

The symptoms begin suddenly either during or shortly after a meal They include epigastric fulness nausea a generalised unpleasant sensation of warmth cold sweats on the face and palpitations There may also be borborygmi and diarrhœa severe muscular weakness drowsiness and syncopal attacks The epigastric fulness is probably due to distension of the jejunum rapid absorption of sugar from the duodenum may cause hyperglycæmic shock and muscular weakness may result from a fall in the blood potassium level

## Duodenal Obstruction

**Etiology** *Acute Duodenal Obstruction* This may occur as part of the mechanism resulting in acute dilatation of the stomach (see p 28)

### Syphilis of the Stomach

A gumma may cause pyloric obstruction, hour glass constriction or ulceration Clinically it simulates peptic ulcer or carcinoma but the Wassermann reaction and the response to anti syphilitic treatment establish the diagnosis Some cases of *linitis plastica* are thought to be syphilitic in origin

Treatment The patient should be given a course of anti syphilitic treatment (see p 596)

## THE INTESTINES

### Duodenal Ulcer

**Definition** A peptic ulcer in the duodenum

**Etiology** The underlying causes are the same as for gastric ulcer (see p 33) A duodenal diathesis is credited by some characterised by a hypertonic hypersecreting and rapidly emptying stomach There is a distinct familial incidence *Predisposing causes* 1 Sex This disease is four times as common in men as in women 2 Age The usual age period is between 30 and 50 years 3 Smoking The effect of this is doubtful as in a series of over 400 cases over 80% were non smokers

**Pathology** *The ulcer is generally situated within an inch of the pylorus* Various stages may be found as with a gastric ulcer The pain is considered to be due to pylorospasm

**Clinical Findings** The patient is usually a middle aged male who gives a history of discomfort later succeeded by pain *in the upper abdomen The pain occurs periodically, lasting for a week or longer* with intervals of relief for several weeks or months These relief periods shorten as the disease progresses The pain is often localised by the finger to a spot in the epigastrium just to the right of the mid line *It may radiate through to the back, near the inferior angle of the right* scapula The pain is related to food in that it occurs about 3 or 4 hours after a meal and tends to wake the patient at about 2 a m It is temporarily relieved by taking more food ("hunger pain") and by alkalis The patient may say that an attack has followed *some dietetic indiscretion* The appetite is usually good but the bowels are constipated In some cases, especially with an acute ulcer melæna hæmatemesis or perforation may be the first indication of a duodenal ulcer

*On Examination* *The patient generally is not wasted* A tender spot may be found in the epigastrium in the subcostal plane about 1½ inches to the right of the mid line The upper part of the right rectus muscle is then somewhat on guard In order to diagnose a duodenal *ulcer further tests are essential* They are —An opaque meal The stomach is usually of the hypertonic type and empties rapidly Irregularity of the duodenal cap may be seen or an ulcer crater in one wall, with a niche due to spasm opposite If there is pyloric stenosis there is delay in stomach emptying *The fractional test meal* There may be no abnormality but if the ulcer is at the hurry stage there

## Jejunal and Gastro jejunal Ulcers

Etiology Jejunal or gastro jejunal ulceration (anastomotic ulcer) occurs as a complication of gastro enterostomy performed for duodenal ulcer in about 1% to 2% of all such operations It is very rare after a gastro enterostomy for gastric ulcer It is met with therefore chiefly in males the important factor being the hyperchlorhydria Other predisposing causes are persistent foci of infection in the nose and throat cranial sinuses gall bladder and appendix and trauma due to pressure of the clamps at the operation or stitches which are not absorbed

**Pathology** The ulcer is usually single it may occur at the site of anastomosis (gastro-jejunal) or in the efferent loop of the jejunum (jejunal) usually within an inch of the anastomosis

**Clinical Findings** The symptoms resemble those of duodenal ulcer They may appear directly after the operation or be delayed for as long as 17 years The pain is usually located to a point just to the left of the umbilicus Examination under X rays with an opaque meal demonstrates a tender spot in the jejunum

**Course and Complications** Perforation may occur with general peritonitis or the formation of a gastro jejuno colic fistula Erosion of an artery may result in severe hæmorrhage

**Prognosis** Jejunal ulcers if untreated do not heal spontaneously There is a tendency for recurrence after medical or surgical treatment

**Treatment** *Prophylactic* Vagotomy or partial gastrectomy combined with pylorectomy or gastro-enterostomy should diminish the incidence of anastomotic ulcer Gastro enterostomy is contra indicated when the gastric acidity is high and the rate of stomach emptying rapid owing to the risk of formation of an anastomotic ulcer In patients over the age of 50 who will not adhere strictly to medical treatment a short-circuiting operation may be performed apart from stenosis This is because the gastric acidity tends to fall after the age of 50

*Curative* A strict course of medical treatment as for gastric ulcer should be tried If this fails operative treatment consists in excision of the jejunal ulcer and undoing the gastro-enterostomy providing that the original duodenal ulcer has healed

## Gastro Colic Fistula

Etiology A gastro-colic fistula may follow the formation of an anastomotic ulcer which has developed after a gastro enterostomy In other cases carcinoma of the stomach may spread directly to the colon

Clinical Findings The principal symptoms are diarrhœa occurring 2 to 3 hours after food and during the night with eructations having a fæcal odour *On Examination* There is usually loss of weight An excess of fat is generally present in the stools the fat splitting being normal Further signs of deficient intestinal absorption are a low blood calcium content and a megalocytic anæmia These findings resemble those of sprue but with gastro colic fistula there is generally hyper

*Chronic Duodenal Obstruction* This may result from —
1 Progressive duodenal *stenosis associated* with an ulcer
2 External pressure, due to adhesions to the gall bladder
3 Visceroptosis which may be confined to the duodenum
The third part of the duodenum is constricted by the root of the *mesentery and the superior mesenteric vessels the first* and second parts of the duodenum dilate, causing chronic duodenal ileus

### Chronic Duodenal Ileus

**Clinical Findings** The patient may experience no symptoms the dilated duodenum being discovered on routine X ray examination In other cases there are periodical attacks of illness characterised by epigastric discomfort gastric flatulence which is succeeded in some cases by severe pain in the epigastrium to the right of the mid line with vomiting of large quantities of bile stained fluid In the intervals between the attacks the patient may feel perfectly well or suffer from malaise and headache The diagnosis is established by a barium meal Cholecystitis may occur as a complication

**Treatment** Medical treatment consists in washing out the duodenum with a duodenal tube If this is not successful a posterior gastro enterostomy or partial duodeno gastrectomy may afford relief

### Duodenal Diverticula

These are occasionally met with They often cause no symptoms and are discovered on X ray examination In some cases persistent symptoms are present, such as epigastric discomfort 1 to 2 hours after food flatulence nausea and vomiting The diverticula occur most often in the second part of the duodenum on its concave side where the vessels enter and there is no peritoneal coat (see diverticulitis p 48)

### Carcinoma of the Duodenum

This is a rare disease It may start near the ampulla of Vater, or extend down *the bile duct to the duodenum from* a primary growth in the gall bladder, or invade the duodenum from a carcinoma of the head of the pancreas

### Duodenitis

(*Proto-duodenitis*)

**Definition** Inflammation of the supra papillary portion of the duodenum

**Etiology** *Duodenitis does not form a definite* clinical syndrome but it may occur as an early stage of a duodenal ulcer or of catarrhal jaundice It may also be associated with a kink at the duodeno jejunal flexure

**Clinical Findings** The patient may complain of pain in the epigastrium relieved by food

*On Examination* Tenderness may be found over the duodenum

**Treatment** This varies with the associated lesions




FIG 2 — COLONIC DIVERTICULA

chlorhydria and in sprue hypochlorhydria The diagnosis is best confirmed by a barium enema

**Treatment** This is surgical for cases due to gastric ulcer, but for those due to malignant disease only symptomatic treatment is available

## Intestinal Diverticula

**Definition** Pouches in the intestine

**Etiology** This is unknown Diverticula occurring in the small intestine may in some instances have a congenital origin but usually they are only recognisable in adult life Meckel s diverticulum is a congenital abnormality of the ileum Diverticula are slightly more common in males, they occur especially after the age of 40 but are not notably associated with obesity

**Pathology** A congenital diverticulum carries with it all the coats of the intestine The acquired variety herniates through the muscular coat, and consists of the mucous membrane and the serous layer, an outer fibrous coat may form In the large intestine the diverticula are often extruded into the appendices epiploicæ so that they are not visible externally until the fat is removed They then appear as bluish black sacs the colour being due to the fæcal contents showing through their thin wall Their necks may be constricted so that the contents cannot escape In the intestine diverticula are found occasionally in the duodenum, rarely in the jejunum and ileum and commonly in the large intestine They are most often present in the pelvic and descending colon very rarely in the ascending colon, but they may occur in the appendix and rectum They are usually multiple in the large intestine

Three stages are described in their formation 1 A prediverticular stage There is localised weakening of the mucous membrane and hypertrophy of the circular muscle of the intestine 2 Diverticulosis Here developed diverticula are present, but they are not inflamed 3 Diverticulitis Inflammation is present in the neck of the diverticulum and in the neighbouring intestine The lumen of the neck is usually obstructed with fæces

**Clinical Findings** A history of constipation is only obtained in about half the cases

*In the prediverticular stage* there are usually no symptoms unless constipation is present X ray examination with an opaque meal or enema shows an area localised to one part of the intestinal wall where the normal haustra are replaced by a series of convex irregularities These areas may be tender on palpation at the X ray examination *With diverticulosis* there may be no symptoms or such symptoms as frequency of micturition, pain in the left lower abdomen, intestinal flatulence, diarrhœa or constipation and discomfort after defæcation may be complained of Abdominal examination yields no information X ray films show the established diverticula which may remain filled after the intestine has emptied (see Fig 2) *When diverticulitis is present* the symptoms are more definite there may be periodical attacks of fever, with leucocytosis and abdominal pain usually in the left lower abdomen

A sausage-like tumour may be palpable in this site The bowels never feel to be properly emptied frequency of micturition may occur especially after defæcation The opaque meal or enema reveals the characteristic appearances The affected portion of intestine is fixed and thickened and spikes may project from the wall like a pallisade These do not alter in position in serial films although the intestine above and below may contract forcibly In about 70% of cases of diverticulitis arthritis of the lumbar vertebræ is found by X rays and there may be severe pain in the back

**Differential Diagnosis** Diverticulitis may be mistaken for left sided appendicitis, actinomycosis of the intestine salpingitis or carcinoma The diagnosis can only be made by a competent radiologist

**Course and Complications** The course is usually slowly progressive Complications include "Pistol shot" perforation in which a stercolith is suddenly forced through the diverticulum Inflammatory perforation with localised or generalised peritonitis Intestinal obstruction Vesical fistula air and fæces appearing in the urine Vaginal or colic fistulæ Carcinoma is a very rare sequela

**Prognosis** This is good in diverticulosis and the early stages of diverticulitis providing adequate treatment is given Death may occur from perforation obstruction or fistula formation

**Treatment** In the acute attacks of diverticulitis the patient should be put to bed rectal injections of 4 to 6 fl oz of warm olive oil are given at night, and a saline colonic washout of 1 to 2 pints of normal saline in the morning The washout is given as follows The saline is warmed to 102° F and run in through a funnel and No 10 soft rubber catheter The catheter is injected for 2 to 3 inches into the rectum with the patient lying in the left lateral position The funnel is held 12 inches above the anus The fluid is run in slowly the patient taking deep breaths The patient is then assisted into the knee-elbow position and after a few minutes lies in the right lateral position He should retain the fluid as long as possible For lesser degrees of diverticulitis and for diverticulosis or the prediverticular stage treatment consists in colonic washouts with saline as above every other day for several weeks and later twice a week for several months These must be adequately given to run round all the colon A low residue diet as for ulcerative colitis should be used and all irritant articles of food avoided Liquid paraffin in doses of m 120 twice or three times a day after meals or I so Gel granules two teaspoonfuls twice a day with meals should be given to keep the motions soft Operation is required for perforation or obstruction

## Regional Ileitis

### (*Crohn s Disease*)

**Definition** A subacute or chronic necrotising and cicatrising inflammation of the terminal segments of the small intestine

**Etiology** Various causes have been suggested These include tuberculosis sarcoidosis amœbic and bacillary dysentery foreign bodies in the intestine the virus of lymphopathia venereum acute mesenteric



FIG 3  SKIP LESIONS IN REGIONAL ILEITIS

may be due to invasion with micro organisms, such as streptococci and the Bact coli rarely with the Streptothrix actinomyces or the Myco bacterium tuberculosis (B tuberculosis) The organisms enter the appendix from the intestine or from a near by inflamed organ in some cases they may be blood borne as from a distant focus in the tonsils or teeth Internal mechanical obstruction from hardened fæces (stercoliths or enteroliths) from pips in food or from thread worms or external obstruction from bands may also lead to appendicitis *Pre disposing causes* 1 Age Chiefly between 10 and 40 years 2 Sex Males predominate slightly 3 Race Especially common in civilised countries In some cases a familial incidence can be traced

**Pathology** The appendix is acutely inflamed with hyperæmia of the mucous membrane and a dull appearance of the peritoneal coat, or it is enlarged and dilated or the mucous membrane is ulcerated The appendix may be filled with pus or it may be gangrenous or perforated with local or general peritonitis In chronic cases it may be distorted and bound down by adhesions and the lymphatic glands at the appendix root and in the ileo cæcal angle enlarged In actinomycosis the appendix and cæcum are frequently involved and the disease spreads to the peritoneum abdominal wall and liver

**Clinical Findings** Acute appendicitis is a surgical complaint and will not be considered in this book Chronic appendicitis frequently causes difficulty in diagnosis It may follow an acute attack of appen dicitis or more often develop insidiously The patient complains of periodical attacks of pain in the right lower abdomen or around the umbilicus of a griping nature (appendicular colic) and there may be nausea vomiting constipation or diarrhœa In the intervals the patient is usually well In other instances he suffers from chronic ill health (appendicular dyspepsia) with such symptoms as recurring nausea anorexia constipation fulness after meals and pains in various parts of the abdomen A momentary sharp stabbing pain may be felt in the right iliac region on walking Frequency of micturition may be noticed if the appendix irritates the bladder A focus of infection in the appendix may be the causative factor in such lesions as gastric or duodenal ulcer cholecystitis or infective arthritis

*On Examination* In a well marked case tenderness can be elicited on firm pressure over McBurney s point (the junction of the outer and middle thirds of a line drawn from the umbilicus to the right anterior superior iliac spine) The pain elicited may be referred to the umbilicus In other cases this tenderness can only be demonstrated when the appendix is filled with barium and palpated while it is visualised with X rays There is often slight rigidity of the right lower rectus muscle Occasionally the appendix lies in an abnormal situation such as in the pelvis when it may be tender on rectal or vaginal examination or it may be retro cæcal or under the right costal margin or even on the left side of the abdomen if the viscera are transposed The temperature is at times slightly raised In a doubtful case inflation of the large intestine through a rectal tube may cause pain in the appendix region

lymphadenitis and trauma None of these suggestions have been substantiated and the cause remains unknown *Predisposing causes* 1 Age The majority of cases occur in the third decade 2 Sex Males are affected more frequently than females

**Pathology** There is a non specific inflammation affecting usually the terminal ileum There is a progressive granulomatous lymphangitis in the intestinal wall and in the mesentery The disease may spread upwards to the jejunum or downwards to the rectum The wall of the ileum is œdematous and thickened the lumen is much reduced and the mucous membrane is partially destroyed Deep ulcers occur between areas of hypertrophied mucous membrane Fistulæ may form between adjacent loops of intestine into the bladder into or around the rectum and through the abdominal wall The latter are becoming much more rare

**Clinical Findings** The disease usually begins insidiously with periodical attacks of diarrhœa and malaise A perirectal or rectal fistula may be the first complaint Abdominal pain usually in the right iliac fossa is present in about 60% of cases The pain is often relieved by defæcation

*On Examination* The fingers are clubbed in about 2% of cases A tender mass may be felt in the right iliac fossa There is often a low grade fever The stools may contain liquid mucus and a little blood but never pus Progressive cases show increasing weakness and psycho neurotic symptoms may be marked In some cases the disease has an acute onset resembling that of acute appendicitis A barium meal shows the characteristic ' string sign ' of Kantor This is a thin irregular linear shadow running from the last filled loop of the ileum through the area of the filling defect to the ileo cæcal valve The disease may spread from area to area of the small intestine leaving normal segments in between In this way the skip lesions and ' skipped areas are produced (see Fig 8)

**Course and Complications** Spontaneous remissions may occur for years but the course is usually progressive the remissions becoming shorter The most important complications are perforation and hæmorrhage

**Differential Diagnosis** A characteristic ' string sign may also be met with in hyperplastic ileo cæcal tuberculosis carcinoid of the ileum endometriosis of the ileum, enterogenous cysts around the ileum or actinomycosis

**Prognosis** Spontaneous cure occurs in about 5% of cases

**Treatment** Medical treatment consists in a low residue diet and the administration of vitamins Sulphonamides penicillin and streptomycin have no specific effect Surgical treatment offers hope of improvement in perhaps 80% of cases but recurrence of the disease is very probable Surgery is necessary in obstruction and with fistula formation

## Appendicitis

**Definition** Inflammation of the appendix.

**Etiology** The cause is not definitely known In some cases it

who complains of passing mucus in the motions She is frequently constipated but there may be periodical attacks of diarrhoea In some cases there are attacks of constipation followed by abdominal colic after which mucus is passed There may also be abdominal discomfort or pain generally in the left iliac region

*On Examination* The patient is usually, but not invariably of a nervous type introspective with her thoughts concentrated on her motions She is thin and the tongue is somewhat furred The descending colon is palpable if it is in spasm or if the patient is constipated and it may be tender The motions are usually constipated and the mucus may be passed as long whitish strips or as a tubular intestinal cast or in balls If the rectum is involved there is frequent tenesmus, with passage of mucus Some blood is at times present in the motions The motions may also contain yellowish brown granules of intestinal sand composed of calcium salts of palmitic stearic and phosphoric acids with urobilin There may be a low grade of pyrexia such as a temperature of 99° to 100° F in the evenings Sigmoidoscopy An excess of mucus is seen covering the mucous membrane no ulceration is present but in some cases the mucous membrane appears inflamed or polypoid

Differential Diagnosis Irritable colon has to be differentiated from colitis associated with carcinoma of the colon or diverticulitis and from ulcerative or catarrhal colitis The diagnosis is established by the history of the mucus in the stools the sigmoidoscopic and barium enema findings the latter showing no evidence of a growth

Course and Complications The disease is very chronic and often is present for many years

Prognosis Cure is often difficult to effect and a relapse is not infrequent after cases have much benefited by a course of rigid treatment

Treatment The patient should be put to bed in severe cases or for the purpose of investigation The patient s anxieties and fears should be relieved as much as possible by a frank discussion of the nature of the illness The constipation must be relieved first by rectal injections of 2 to 4 fl oz of warm olive oil which are later increased up to 10 fl oz This is retained during the night and followed by an enema of warm normal saline in the morning Liq paraffin m 60 to 240 is also given t i d p c Intestinal spasm is combated by a mixture containing Tnc belladon m 5 to 10 sod brom gr 5 sod bicarb gr 10 sp chlorof m 7 aquam ad fl oz ½ Fl oz ½ t d s a c

The diet must not be irritating no foods leaving residue should be eaten no salads skins of fruit or fish or pips must be taken

## Colitis

Definition The term colitis includes inflammation and degeneration of the mucous membrane of the colon and some conditions associated with excessive secretion of mucus The following varieties are described Acute catarrhal Chronic catarrhal Ulcerative

(Bastedo's sign) Ulceration of the mucous membrane of the appendix will account for the presence of occult blood in the fæces

**Differential Diagnosis** Chronic appendicitis must be differentiated *from* intestinal colic, intestinal angio neurotic œdema, pyelitis, a gastric or duodenal ulcer chronic cholecystitis, biliary colic renal colic, a growth near the cæcum, ileo cæcal tuberculosis, regional ileitis, chronic salpingitis or oöphoritis or enlarged glands (usually tuber culous) in the right iliac fossa The urine should always be examined microscopically to exclude the presence of organisms An X ray examination of the abdomen will help to exclude a stone in the gall bladder or kidney or calcified abdominal glands An opaque meal helps to eliminate the possibility of a gastric or duodenal ulcer, and a tender appendix may be palpated under the screen as mentioned above If the appendix fills with barium it is usually possible to deter mine whether or not it is fixed by adhesions if the appendix does not fill, there is evidence that the lumen is occluded and further ileal or cæcal stasis is suggestive of chronic appendicitis A cholecystogram helps to exclude cholecystitis It is often impossible to diagnose ileo-cæcal tuberculosis without an operation but in some cases pulmonary tuberculosis is also present A lump may be palpable on abdominal examination which can be mistaken for carcinoma

**Course and Complications** Chronic appendicitis is characterised *by recurrent attacks of pain or dyspepsia ultimately an attack of* acute appendicitis may develop In other cases the attacks gradually cease, perhaps owing to permanent obliteration of the lumen Com plications include Abdominal adhesions, perforation peritonitis gastric or duodenal ulceration cholecystitis and colitis

**Prognosis** This is always uncertain

**Treatment** A chronically inflamed appendix which is causing symptoms should be removed surgically unless an operation is contra indicated by some coexistent disease Appendicular colic may some times be relieved by the administration of Tnc belladon m 10 to 15 t d s a c The treatment of actinomycosis is described on p 608

### *Irritable Colon*

*(Muco membranous Colic Colon Spasm Muco membranous Colitis Mucous Colitis)*

**Etiology** In many cases irritable colon is considered to be a psycho somatic disease in which as the result of an anxiety state there is inco ordination of colonic movements In some instances there may be an allergic basis others follow the prolonged use of cathartics or enemata In one of my cases it resulted from eating coarse bread *Predisposing causes* 1 Age Usually over 20 2 Sex Females predominate 3 Nervous instability is often present

**Pathology** Often no changes are found post mortem in the mucous membrane of the colon, but a true inflammatory or granular colitis may be present

**Clinical Findings** The patient is usually a middle aged woman

up to 10 or 20 times in the 24 hours Sigmoidoscopy reveals a swollen and red mucous membrane with a mucous exudate, superficial ulcers may be seen especially on swabbing away the mucus In advanced stages X ray examination with a barium enema shows the colon as a rigid narrow tube the haustrations usually being absent

**Differential Diagnosis** Ulcerative colitis must be differentiated from a growth in the colon from mucous colitis dysentery and pernicious anæmia If there is a growth it may be seen with the sigmoidoscope or if situated higher up mucus and blood may be seen coming down the colon the lower part of the mucous membrane appearing healthy In chronic dysentery the bacilli can usually be isolated from a swab taken from an ulcer through the sigmoidoscope The blood count serves to differentiate pernicious anæmia

**Course and Complications** The disease tends to run a very chronic course Complications include polyposis or stricture of the colon peritonitis and arthritis In some cases carcinoma may develop in the colon

**Prognosis** This is usually grave prolonged treatment for months or years is necessary to effect arrest of the disease and even then relapse is liable to occur

**Treatment** The patient should be put to bed and an enquiry made concerning psychogenic causes An understanding physician may do much good by tactful reassurance and encouragement The diet should be of a low residue type I have found the following suitable —

*Breakfast* Weak tea or coffee with milk A choice of Porridge (strained) with milk crisp toast (well chewed) with butter egg and lean bacon white fish (grilled baked or steamed) pipless jam jelly marmalade honey

*Lunch* A choice of Cream of vegetable soup lean tender meat rabbit chicken sweetbreads brains kidneys white fish (grilled baked or steamed) mashed or boiled potatoes purée of vegetables milk pudding fruit purée jelly fruit juices ripe bananas mild cheese toast butter cream

*Tea* Tea with milk Crustless bread and butter spongecake or plain cake pipless jam honey or syrup

*Supper* As for lunch

Sugar may be taken as desired On waking at 11 a m and last thing at night milk flavoured as desired

*Articles of food to be avoided* Fried food pastry suet pudding made up meat or fish dishes coarse bread or biscuits strong tea or coffee raw fruit salads pickles nuts

Phthalyl sulphathiazole or sulphathalidine G 1 t i d for 14 days should be given a trial and if penicillin sensitive organisms are found in the fæces subsequently a course of intramuscular injections of penicillin may be helpful The dose is 300 000 units of a *procaine penicillin* morning and evening for 5 to 7 days A starch and opium enema (starch gr 60 water fl oz 2 and tinc opii m 30) should be given daily until the motions are reduced to about four a day Rectal injections of cod liver oil may then be given using first fl oz 2 every morning and

## Acute Catarrhal Colitis

*Etiology* Acute catarrhal colitis may occur as a part of acute gastro enteritis (see p 24), sometimes due to an infection with the Bacterium friedlanderi (pneumobacillus) to influenza or to drugs such as colchicum or it may be associated with such diseases as enteric fever intestinal tuberculosis, dysentery an intestinal neoplasm or uræmia

**Clinical Findings** The patient complains of diarrhœa, and the *stools contain mucus and at times bright blood* There is usually some abdominal colic and the *temperature may be raised*

**Treatment** This varies with the cause, and is as described under the various diseases with which it is associated

## Chronic Catarrhal Colitis

**Etiology** Chronic catarrhal colitis may be a sequela of acute catarrhal colitis, or of dysentery, or it may result from the misguided habitual use of purgatives

**Clinical Findings** The motions are loose frequent and contain some bright blood and mucus

**Treatment** This again varies with the cause In general a diet should be taken which is free from irritants (see p 55) Liquid paraffin m 60 should be taken t d s p c, and if there is pain due to *spasm of the colon a mixture containing Tnc belladon m 7 to 10 t d s* a c usually affords relief An abdominal belt should be worn as the condition is made worse by chill

## Ulcerative Colitis

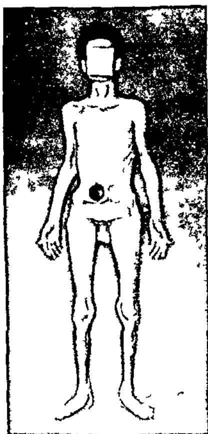
**Definition** A chronic affection of the colon characterised by ulceration of its mucous membrane Ulceration due to dysentery, tuberculosis syphilis and enteric fever is not included under *this* heading

**Etiology** The cause is not known by some it is believed to be due to dysentery or pseudo dysentery bacilli or to some undiscovered infective agent In some cases psychological factors appear to be of importance and stress has been laid on the dependent personality of the *patient*

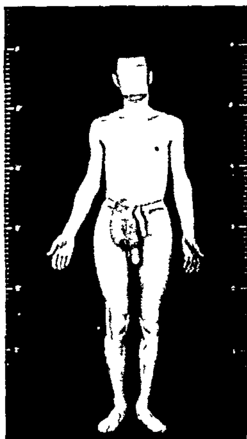
**Pathology** The ulcers are superficial and chiefly situated in the pelvic colon

**Clinical Findings** The patient is usually an adult who complains of diarrhœa which gradually becomes worse The onset may be quite sudden *without any emotional disturbance or it may follow* a period of worry or be associated with over conscientiousness in work There is often abdominal discomfort or pain in the left iliac region If the lesions are low down there is also tenesmus

*On Examination* The patient is usually wasted and *anæmic* and there may be a low grade pyrexia of 99° to 100 F The sedimentation rate of the red cells is usually increased The stools are loose and contain mucus bright blood and at times some pus They may occur



A



B

Fig 4 Patient suffering from Ulcerative Colitis showing Condition A 10 Days after Ileostomy and Subtotal Colectomy Weight 5 st 13 lb and B 7 Months later Weight 10 st

increasing to fl oz 8 When the patient has become accustomed to retain them for several hours the injections should be given in the evening Vitamin B should be administered as Beplex capsules two daily and ascorbic acid mg 50 t i d *For the anæmia blood transfusions* of 2 pints are of the greatest value, and should be repeated until the hæmoglobin level reaches 80 to 90% Iron should then be given in doses of gr 30 to 60 t i d of ferri et ammon cit providing it does not increase the diarrhœa In some cases the barium meal reveals marked intestinal hurry, and this is an indication that tincture of belladonna should be given by mouth in doses up to m 10 to 15 three or four times a day with an injection of atropine sulphate gr 1/75 last thing at night The dosage must be regulated by the patient s tolerance as judged by the effect on accommodation or the appearance of a rash Phenobarbitone gr ½ two or three times a day is also helpful or codeine gr ½ twice daily if there is much pain In addition to the lines of treatment detailed above, cortisone may be given by mouth in doses of mg 100 daily for 6 weeks In a therapeutic trial with controls given an inert tablet, the cortisone treated patients, at every stage of the severity of the illness, and in both first attacks and relapses did better than the corresponding control patients Unfortunately the effect of the cortisone is not permanent If these measures fail surgical treat ment should be considered This should not be delayed until the patient is very debilitated Several varieties of operation have been recom mended but a terminal ileostomy is the operation of choice and this *in the majority of cases has to be permanent the patient wearing a* Koenig Rutzen bag This is usually followed by colectomy The good result which may ensue from these operations is illustrated in Fig 4 The mortality rate for the operation varies between 10 and 20%

## Intussusception

**Definition** Telescoping of the intestine There are two clinical varieties Acute and chronic

### Acute Intussusception

**Etiology** Acute intussusception results from irritation of the intestine and muscular imbalance in its walls The irritation may be due to hard fæces worms or a polypus *Predisposing causes* 1 Age Usually infants 2 Sex Males predominate

**Pathology** The upper portion of the intestine is invaginated into that below There are three layers, the inner, entering or intus susceptum the returning or middle and the outer ensheathing or *intussuscipiens* *The mesentery enters with the intestine* and com pression of the vessels may lead to inflammation gangrene or rupture of the intestine There are four anatomical varieties 1 The ileo cæcal This is the most common the ileo cæcal valve and the ileum enter the colon 2 The enteric Here one portion of small intestine enters another 3 The colic The colon is invaginated 4 The ileo colic The ileum passes through the ileo cæcal valve and then the ileum ileo cæcal valve and cæcum pass into the colon

**Clinical Findings** The infant is suddenly taken ill with abdominal colic. He may then vomit. The characteristic feature is the loose actions of the bowels there is tenesmus and passage of odourless mucus and blood.

*On Examination* In the ileo-cæcal variety a ' sausage shaped " tumour may be felt in the upper part of the abdomen or in the left iliac region whereas the right iliac region feels empty (*signe de Dance*). The temperature falls but the pulse is frequent.

**Differential Diagnosis** Acute intussusception is most likely to be confused with Henoch s purpura (see p 547). Careful search must be made for purpuric spots in the skin. If found no operation must be performed. In acute colitis the motions have a fæcal odour and no tumour is palpable.

**Course and Complications** Collapse and death ensue unless the intussusception is reduced.

**Prognosis** This depends upon efficient treatment.

**Treatment** This is surgical.

## Chronic Intussusception

**Etiology** Chronic intussusception occurs in adults and is usually associated with a simple or malignant growth of the large intestine.

**Clinical Findings** The patient suffers from attacks of abdominal colic with diarrhœa the passage of blood and mucus and at times vomiting.

*On Examination* A tumour is palpable in the abdomen in some cases or the invaginated intestine may even protrude from the anus.

**Differential Diagnosis** A barium enema may disclose the nature of the trouble.

**Course and Complications** The intussusception may unravel itself spontaneously but usually it tends to recur and after persisting for several months results in obstruction or perforation.

**Prognosis** The outlook is unfavourable.

**Treatment** This is operative.

## Intestinal Obstruction

**Definition** Obstruction to the passage of fæces through the intestine.

**Etiology** The obstruction may be due to 1 *Causes in the lumen or wall of the bowel*. These include —A volvulus. An intussusception. A growth. Impacted fæces. A stricture due to syphilis or following dysentery. A foreign body such as a gall stone which has ulcerated through into the duodenum (the obstruction then usually occurs in the terminal part of the ileum). Other foreign substances such as masses of thread worms or a hair ball. Paralytic ileus in which there is no mechanical obstruction this may follow an abdominal operation or complicate acute appendicitis peritonitis or torsion of the pedicle of the spleen. Regional ileitis (Crohn s disease) in which there is cicatricial stenosis usually of the last 12 to 14 inches of the ileum following a chronic inflammatory condition of unknown origin. Multiple fistulæ

obstruction A direct X ray of the abdomen will show excess of gas in the large intestine if this is obstructed The presence of gas in the small intestine is also a sign of obstruction there and in some cases fluid levels are seen

**Differential Diagnosis** Intestinal obstruction must be diagnosed from other acute abdominal lesions such as a perforated gastric or duodenal ulcer acute appendicitis mesenteric thrombosis and acute pancreatitis and from such conditions as biliary or renal colic, lead colic and abdominal crises in tabes All the hernial orifices should be examined The history of the case the results of the clinical examination and the inability of the patient to pass fæces or flatus usually enable the diagnosis to be established before the stage of fæcal vomiting is reached

**Course and Complications** Unless relieved surgically intestinal obstruction is usually permanent. Complications such as perforation and peritonitis may occur

**Prognosis** Death occurs in a few days, unless the obstruction is relieved

**Treatment** An operation should be performed without delay to relieve the obstruction if it is not paralytic in origin Morphine should not be given until the diagnosis has been made For paralytic ileus stimulant treatment is first applied One ml of Pituitrin is injected intramuscularly followed in about a quarter of an hour by a glycerin enema (glycerin and water of each fl oz ½) If no flatus is passed further stimulant treatment should be applied such as the intramuscular injection of a mixture of acetyl choline 0 2 G Pituitrin 0 5 ml and physostigmine salicylate gr 1/64 every hour for four doses This is followed by an enema of ox bile fl oz 2 and normal saline fl oz 4 a simple enema being given half an hour later If this fails it is best to abandon stimulant treatment and inject subcutaneously morphin sulph gr 1/6, apply heat to the abdomen by means of an electric cradle and give an intravenous drip injection of 1 pint of normal saline containing 5% dextrose The electrolytic balance of the body fluids should be maintained the amount of sodium potassium calcium or bicarbonate required being determined by blood estimations To relieve vomiting a Ryle s stomach tube should be passed and suction applied either by a syringe or by attaching the tube to an inverted bottle containing water suspended above the patient whereby syphonage can be established *Acute distension of the small intestine can be relieved by a Miller* Abbott double lumen tube with an inflatable rubber balloon situated near to the perforated metal tip

### Intestinal New Growths

**Etiology** The cause of new growths is not known Carcinoma is usually a disease of adult life being very uncommon in infants and children The sexes are equally affected *Varieties* An intestinal tumour may be simple such as an adenoma polypus myoma lipoma or hæmangioma or more commonly a malignant tumour such as a carcinoma Sarcoma is rare

may be present communicating either with the large intestine, or tracking through the anterior abdominal wall

2 *Causes outside the bowel* Strangulation or obstruction may result from —1 A band This may be due to plastic peritonitis or follow a laparotomy The band may form between two portions of the mesentery or between the mesentery and an abdominal viscus or inflamed gland 2 A cord This may pass from the omentum to an abdominal viscus or to the abdominal wall 3 Meckel's diverticulum may be attached to the umbilicus or to some viscus in the abdomen or to the mesentery 4 The bowel may be strangulated or obstructed in an internal hernial orifice, in the foramen of Winslow, or in a diaphragmatic hernia

In strangulation the flow of blood through the vessels of the bowel is interfered with but the passage of fæces is not prevented

**Pathology** The portion of bowel above the obstruction is dilated the wall is hyperæmic and the contents are fluid consisting of an exudate from the wall and vessels and many bacteria Histamine may form in the contents and on absorption give rise to the symptoms of shock At the site of the block the intestine is also distended its coat becomes purplish in colour and it may be gangrenous and sloughing with the vessels thrombosed It usually contains blood stained fluid and gas Below the obstruction the bowel is empty contracted and pale Peritonitis may occur around the site of the obstruction The obstruction may occur acutely as with a band, volvulus intussusception or incarceration in a hernial orifice or more gradually, as with a growth in the large intestine where hardened fæces may prove the last factor producing a total block The ' fæcal vomiting is probably not due to antiperistalsis and regurgitation of fæces but to an increased exudate into the bowel the fluid contents well up to the stomach, and are vomited without effort

**Clinical Findings** With acute obstruction the patient is suddenly taken ill with severe tearing and colicky pains in the abdomen which double him up, he becomes prostrated and collapsed Vomiting sets in and there is great thirst The vomit at first consists of the gastric contents later of bile and finally it is of the fæcal regurgitant type A motion may be passed early in the attack, but after this no fæces or flatus escape from the rectum

*On Examination* The patient is seen to be very ill he is pale the skin is moist the tongue furred and later dry, the temperature is sub normal and the pulse frequent The abdomen is generally distended but it is not always tender A tumour may be felt as with a volvulus or visible peristalsis may occur from time to time The higher up in the intestine that the obstruction occurs the more acute are the symptoms The blood urea and non protein nitrogen figures are raised In chronic obstruction the onset is more gradual and diarrhœa may at first alter nate with constipation then absolute constipation with symptoms of obstruction sets in The patient should be given a turpentine enema (turpentine fl oz ½ and soap and water fl oz 10) This is returned clear without any force and without the passage of flatus in the presence of

metastases have formed Death usually occurs in 1 to 2 years from the appearance of symptoms

Treatment In cases of hæmangioma the affected portion of the intestine should be resected Simple tumours may be removed surgically and malignant ones either treated by operation or by radiotherapy A colostomy may be required as a palliative measure to relieve obstruction

### Intestinal Argentaffin Carcinoma

*(Malignant Carcinoid of Intestine Carcinoidosis)*

Definition A carcinoma the granules of the cells having an affinity for silver salts

Pathology Malignant carcinoid tumours usually arise in the ileum Secondary deposits occur in the liver skin kidneys adrenals lungs and brain A substance 5 hydroxytryptamine (5 HT) also known as serotonin is produced in the argentaffin cells of the tumour and the secondary deposits It is liberated in the blood and some is taken up by the platelets The spleen is particularly rich in it 5 HT is probably a hormone and is normally produced in the argentaffin cells of the gastro intestinal mucosa It causes contraction of plain muscle but when injected into an animal it produces vasodilatation It has also an anti diuretic effect It is inactivated in its passage through the lungs and is excreted in the urine as a breakdown product 5 hydroxyindole acetic acid (5 H I A A )

Clinical Findings When a large amount of 5 HT is being produced especially when there are hepatic metastases the patient exhibits a peculiar syndrome characterised by cyanosis attacks of flushing dyspnœa and diarrhœa In addition there may be evidence as shown by X rays of a lesion in the intestine with an enlarged liver and ascites The tumour may cause abdominal pain or symptoms of obstruction Another peculiar feature of the syndrome is the presence of pulmonary stenosis and there may be tricuspid regurgitation Nodules may be present in the skin

Differential Diagnosis This may be established by microscopical examination of a nodule in the skin and by finding an increased excretion of 5 H I A A in the urine Mistakes may be made in diagnosing the condition as asthma right sided heart failure cirrhosis of the liver sprue or dysentery or the flushes may be considered to be due to the menopause

Treatment As the tumour is slowly growing resection of the primary growth even if secondary deposits are present may be worth while

### Hirschsprung's Disease

*(Congenital Idiopathic Dilatation of the Colon Megacolon)*

Definition Dilatation and hypertrophy of the colon arising without obvious organic obstruction

Etiology It is considered to be a congenital anomaly of the pelvic parasympathetic system due to absence of ganglion cells in Auerbach s plexus in a variable portion of the distal colon This results in failure

**Pathology** Polypi may be multiple occurring in the large intestine Diffuse polyposis of the colon of the hereditary type differs from post inflammatory polyposis Malignant degeneration almost invariably occurs in the former if the patient lives long enough *Hæmangiomata* also may be multiple Carcinoma is found in the large intestine in the following sites in order of frequency The rectum pelvic colon, cæcum transverse colon *splenic flexure ascending colon* hepatic flexure and descending colon It is rarely met with in the appendix and small intestine Secondary deposits occur comparatively late in the disease

**Clinical Findings** Hæmangioma of the small intestine may give rise to recurrent attacks of melæna with severe anæmia but usually no *pain* *The patient suffering from a malignant growth is usually an adult* over the age of 40 The first symptom may be the onset of persistent constipation or of periodical attacks of diarrhœa or of alternating *constipation and diarrhœa, where the bowels previously have acted very* regularly The patient may also notice abdominal discomfort or fulness a feeling of general illness and loss of weight The appetite may remain good *He may complain of bleeding from the rectum or of a frequent* desire to go to stool if the growth is low down in the rectum

*On Examination* In the early stages often no localising signs can be found as the growth enlarges it may become palpable on abdominal or rectal examination Some pallor and cachexia may also be apparent There may be slight and irregular fever The motions may contain macroscopic or occult blood and mucus or pus In some cases ribbon shaped motions are passed due either to compression by the growth or to reflex anal spasm With the sigmoidoscope the lower 10 inches of the alimentary tract can be inspected and this examination should never be omitted in a doubtful case A barium enema is more likely to reveal a growth in the large intestine than is a barium meal In more advanced cases evidence of secondary deposits may be found in other organs such as the liver and there may be ascites

**Differential Diagnosis** Hæmangioma is not often diagnosed even at laparotomy It should always be borne in mind in cases of recurrent melæna and anæmia Telangiectases may be seen in the serous coat of the affected part of the intestine The diagnosis of an intestinal malignant growth depends upon the combined results of a *manual* examination sigmoidoscopy the barium enema and the occult blood test Other conditions which require exclusion are Hæmorrhoids simple constipation redundant loops of the colon colitis *diverticulitis* actinomycosis Crohn s disease a chronic appendix abscess a gastric neoplasm enlargement of the spleen, enlargement of the gall bladder or kidney, hyperplastic tuberculosis of the cæcum *tuberculous peritonitis* and ascites due to other causes

**Course and Complications** The course is slowly progressive secondary deposits occur in *the abdominal glands peritoneum* and other organs, such as the liver and brain Intestinal obstruction or perforation may occur as complications

**Prognosis** *This is fatal unless the growth* can be removed before

shown by frozen sections at the operation the proximal bowel being joined to the anus with preservation of the sphincter The operation is preceded first by bowel washouts and then by a right sided colotomy Finally the colotomy is closed

## Steatorrhœa

The passage of loose pale fatty stools may be due to disease or disorder of the pancreas the liver or the intestines

*Pancreatic Steatorrhœa* This may result from chronic pancreatitis fibrocystic disease pancreatic calculi or disturbance of function due to hypoprotinæmia caused by malnutrition The diminished secretion of pancreatic enzymes causes delayed absorption of carbohydrates proteins and fats from the intestine Duodenal intubation shows diminution or absence of pancreatic ferments

*Hepatic Steatorrhœa* This is due to an absence or diminished amount of bile salts in the intestine The stools are usually well formed but very fatty

*Intestinal Steatorrhœa* Grossly abnormal changes may be present such as regional ileitis Whipple s disease tuberculosis and scleroderma A blind loop in the intestine following a short circuit operation may result in severe ill health and steatorrhœa possibly owing to alteration in the intestinal flora Gastro colic fistula also causes steatorrhœa and it may result from gastro enterostomy

*Whipple s disease* or intestinal lipodystrophy is characterised by abdominal discomfort fatty diarrhœa loss of weight and in some cases arthralgia brown pigmentation of the skin and buccal mucous mem brane with low blood pressure At autopsy the intestinal villi are found to be filled with fat and foaming macrophages containing glycoprotein are present in the mesenteric gland sinuses

*Idiopathic Steatorrhœa* Under this heading are included certain conditions such as cœliac disease tropical sprue and non tropical sprue resulting in diminished absorption from the intestine without any gross structural lesions being present In some cases a rapid intestinal transit time the barium meal reaching the rectum in two hours will cause a fatty diarrhœa

## Cœliac Disease

(*Gee s Disease Gee Herter Disease Pancreatic Infantilism Intestinal Infantilism*)

**Definition** A disease of infants characterised by difficulty in the assimilation of fat wasting and retardation of development

**Etiology** It has been shown in Holland and England to be due to wheat gluten in the diet in susceptible children It is not known whether all children suffering from cœliac disease are intolerant of gluten *Predisposing causes* 1 Artificial feeding 2 Age 1 to 5 years 3 Sex Girls predominate

**Pathology** There is inability to assimilate fat and carbohydrate Post mortem a round celled infiltration may be found in the intestinal mucous membrane and in some cases perilobular pancreatic fibrosis

of normal peristalsis to pass along the affected portion of intestine with dilatation of the colon above The cases which manifest themselves in adult life (megacolon) may have a similar origin but are aggravated by constipation Some cases occur in association with steatorrhœa (see p 63) The disease is more common in males than in females

**Pathology** The descending and pelvic colon are chiefly affected but the rectum is involved in megacolon *The colon is much enlarged* and may measure 12 inches in diameter, the circular and longitudinal muscular coats are hypertrophied and stercoral ulceration of the mucous membrane may be seen *In a severe case the contents of the colon* may weigh over 3 stones In Hirschsprung s disease there is a narrow terminal segment of aganglionic intestine in the rectum and recto *sigmoid area with dilated colon above it* *In acquired megacolon* (pseudo Hirschsprung s disease) the dilatation of the colon extends down to the anal region In Hirschsprung s disease the parasympathetic *ganglion cells are completely absent in the narrow segment of the colon*

**Clinical Findings** The condition may be noticed soon after birth the infant being extremely constipated and the abdomen becoming distended In other cases the distension is not apparent until adult life The patient further complains of ill health occasional vomiting abdominal colic and some dyspnœa from upward displacement of the diaphragm The bowels may not be opened for several months but attacks of diarrhœa sometimes occur

*On Examination* The abdomen may present a ballooned appearance being either diffusely distended or more so on the left side Visible peristalsis may be present Digital examination shows the rectum loaded with fæces in acquired megacolon and there is often some anal abnormality such as a painful fissure or congenital narrowing In Hirschsprung s disease the anal canal is normal and the rectum is empty of fæces A barium enema may reveal an enormously distended colon and a narrowed distal segment in Hirschsprung s disease

**Differential Diagnosis** Other causes of severe chronic constipation must be excluded such as a stricture or a growth Usually there is no difficulty in establishing the correct diagnosis

**Course and Complications** The course is progressive unless relieved by treatment Complications include perforation of the colon volvulus or complete intestinal obstruction

**Prognosis** Children thus affected do not usually survive to adult life, if untreated Death may occur from obstruction perforation starvation or intercurrent infection

**Treatment** An attempt should be made to relieve the constipation by enemata if the rectum is involved it may be emptied digitally or with an instrument such as a spoon and *the anal sphincter dilated daily* by a conical bougie The colon should then be washed out daily Spinal anæsthesia effective up to the 5th Th root may immediately result in the bowels being opened *This treatment may be repeated in a few* weeks This is often successful in mild cases of Hirschsprung s disease In severe cases with a long and narrow distal segment good results may be obtained by *excision of all the aganglionic portion of colon* as



Fig A Child suffering from Cœliac Disease Age –C Months Weight 18 Lbs 4 Ozs Height 0½ Inches

[T f p 64

**Clinical Findings** The onset is insidious the infant fails to gain in weight and is backward in walking the appetite is poor and there is looseness of the bowels

*On Examination* The child looks younger than its years, the height is less than normal, there is loss of the subcutaneous fat, wasting especially of the buttocks and limbs, and sexual immaturity The face is not necessarily thin but the skin may be pigmented The abdomen is swollen with a flatulent distention (see Fig 5) The liver and spleen are not enlarged The urine is normal The bowels are opened 4 or 5 times in the 24 hours, the motions being bulky, pale, frothy and offensive They contain an excess of fat with an average of about 50% fat in the dried fæces of which the greater part is composed of fatty acids There is also a deficiency of bile pigment A non-diarrhœic type is also described in which all the symptoms are less severe X ray examination of the bones shows delay in ossification or rachitic changes The oral dextrose tolerance curve and the vitamin A absorption curve are *typically flat* and the barium meal shows a clumping of the barium and a loss of the normal feathering appearance of the small intestine The trypsin content of the aspirated duodenal juice is normal

**Differential Diagnosis** The clinical picture is characteristic Other conditions which require exclusion are Hirschsprung s disease, tuberculosis of the mesenteric glands causing obstruction of the lacteals steatorrhœa sprue and fibrocystic disease of the pancreas In Hirschsprung s disease constipation has usually been noted shortly after birth fatty stools may occur with lacteal obstruction in sprue the appetite is usually good and the tongue is sore and in pancreatic steatorrhœa *there is no interference with growth, and the fæcal fat is chiefly unsplit* In fibrocystic disease of the pancreas (see p 75) cystic or bronchiectatic changes may be found in the lungs the fingers may be clubbed and trypsin is absent, or only present in small amounts in the duodenal contents

**Course and Complications** The course is usually prolonged unless adequately treated Complications such as rickets a generalised but slight œdema tetany or purpura may occur

**Prognosis** There is a tendency to recovery in the course of 2 or 3 years but death may result from an intercurrent disease The immediate outlook has been much improved by the introduction of a *gluten free diet* In severe cases remarkable improvement may be obtained in a period of 2 to 3 months The expectation of normal development as regards height and sexual maturity is always uncertain Follow up studies of young adults who had cœliac disease in childhood show there is a tendency to relapse

**Treatment** The diet must be gluten free as gluten inhibits fat absorption in cœliac disease Wheat flour and rye flour contain gluten If gluten is removed from wheat flour wheat starch is well tolerated and there is no need to restrict carbohydrate and fat In severe cases the initial diet consists of skimmed milk Prosol (1 drachm of powder in 1 oz of water) glucose and banana purée The diet is increased by adding minced chicken egg custard biscuits made from soya flour

butter sieved vegetables potatoes cornflour, etc Ascorbic acid mg 25, halibut liver oil 10 drops and tab Benerva co 1 t i d should be given daily For anæmia iron should be given by mouth or a blood trans fusion may be required It may be necessary to keep the child on a gluten free diet during the years of growth If the child is put back on a normal diet and the growth and weight fall off the special diet must be given again

### Cœliac Disease in Adults

*(Idiopathic Steatorrhœa Gee s Disease Non tropical Sprue Gee Thaysen Disease)*

**Definition** A disease of adults characterised by steatorrhœa and disturbances of calcium and phosphorus metabolism

**Etiology** The cause is unknown Some patients are intolerant of gluten *Predisposing causes* 1 Age Adolescents and adults 2 Sex Females predominate slightly

**Pathology** Post mortem examination shows no characteristic change

**Clinical Findings** A history can often be obtained of diarrhœa or of rickets in infancy The patient complains of such symptoms as diarrhœa weakness skin eruptions muscular cramps pains in the bones or bony deformities especially knock knee

*On Examination* The growth is stunted and the abdomen is often protuberant The patient presents an infantile appearance Skin lesions may be seen such as areas of erythema pigmentation or exfolia tive dermatitis especially on covered portions of the skin Spontaneous fractures may be found in various bones genu valgum or varum is common and the bones show osteoporosis The fingers may show parrot beak clubbing In some cases the colon is distended (megacolon) Changes in the small intestine have also been found by X ray examina tion such as obliteration of the markings of the valvulæ conniventes distention of the gut and segmentation of the contained barium into clumps The blood There is often an anæmia either hypochromic megalocytic or erythroblastic in type The serum calcium is often low the plasma phosphorus is usually low but may be normal or high and the alkaline phosphatase content rises with the activity of the bony changes The blood sugar is often low and the sugar tolerance curve flat The stools are either fatty or appear normal to the naked eye but chemical examination reveals an excess of fat (40 to 70%) The fats are well split Increased muscular irritability may be demonstrated by Chvostek s or Trousseau s sign (see p 694) or fully developed attacks of tetany may occur Opacities in the lens are revealed by the slit lamp in some cases

**Differential Diagnosis** Cutaneous diseases such as psoriasis or exfoliative dermatitis may be diagnosed without the true nature of the disease being recognised In other instances tetany is the predomi nating feature The clinical findings resemble those of sprue (see p 728)

**Course and Complications** The course is usually progressive unless

**Rectal Constipation or Dyschezia** This may be due to —

1 Bad habits An attempt to have the bowels opened is not made daily at some fixed hour or the desire to defæcate is neglected

2 Loss of muscle tone This may result from habitual neglect to the call or it may be due to a nervous lesion such as transverse myelitis The perineal muscles may be weakened by childbirth and the abdominal muscles by lack of exercise

3 Pain as that due to a fissure or to piles

4 Obstruction, from a growth in the rectum a stricture or a pelvic tumour

**Clinical Findings** Chronic constipation is a common complaint which often causes no symptoms The bowels may be opened daily but evacuation is incomplete In such cases the patient is not aware that he is constipated Normally the bowel should be emptied up to the splenic flexure In other cases the individual may not be constipated although the bowels only move every other day Constipation is sometimes accompanied by vague symptoms of ill health such as a muddy complexion furred tongue anorexia headache lassitude flatulence abdominal discomfort or colic The diagnosis of the type of constipation can only be made with certainty by means of a barium meal In dyschezia a digital rectal examination reveals the presence of fæces even directly after the bowels have been opened Sigmoidoscopy should always be made in a doubtful case to exclude the presence of a growth in the rectum

**Differential Diagnosis** The barium meal will show where the delay occurs normally the meal should reach the cæcum in 4½ hours, the hepatic flexure in 6½ hours the splenic flexure in 9 hours the pelvic colon in 12 hours and the rectum in 18 hours

**Course and Complications** The course is usually chronic the constipation tending to become more severe unless adequately treated Complications such as colitis stercoral ulceration of the colon a fissure or hæmorrhoids may ensue

**Prognosis** This must depend upon the cause of the constipation and the possibility of effective treatment

**Treatment** *Prophylactic* Regular habits should be encouraged in children and adults The dietary should be well balanced and contain sufficient fluids

*Curative* Habits The patient should attempt to have the bowels open every morning after breakfast in dyschezia a squatting position should be assumed Exercise General exercises local abdominal and perineal exercises and abdominal massage are all of value Diet The diet should include stewed fruit such as prunes fresh fruit dates nuts green vegetables root vegetables salads wholemeal bread porridge treacle coffee and butter cream or olive oil

Medicines For dyschezia the bowels may be emptied at the beginning of the treatment by a glycerin suppository and later by daily saline colonic washouts using 1 to 1½ pints of warm normal saline run in at a pressure of 18 inches the volume being gradually reduced (see p 49) If the fæces are very hard they may be softened

adequately treated Intercurrent infections, such as broncho pneumonia may ensue or hæmorrhages from mucous membranes into the skin or joints or from the genito urinary tract due to vitamin K deficiency

**Prognosis** The welfare of the patient can be very materially improved by adequate treatment

**Treatment** The diet must be free from wheat flour and rye flour which contain gluten A diet such as that described above for cœliac disease in infants is suitable Calcium should be administered in the form of calcium lactate gr 40 t i d and vitamins A $B_1$ $B_2$ C and D should also be given Suitable preparations are Vitamins A and D Adexolin capsule (liq vitamin A et D conc) m 3 or caps vitamin A et D (N F) 1 t d s $B_1$ Benerva tab (aneurin hydro chlor B P) mg 3 1 daily $B_2$ riboflavin tab mg 10 1 daily and C Redoxon tab (ascorbic acid tab) mg 50 1 daily In tetany calcium should be administered as described on p 695 Hypochromic anæmia and erythroblastic anæmia should be treated with iron such as ferri et ammon cit gr 15 to 30 t d s and megalocytic anæmia with Marmite ½ oz daily or the intramuscular injection of liver Two ml of a crude liver extract such as Hepastab or Livadex should be given every other day This is also thought to aid fat absorption

## Constipation

**Definition** Delay in the passage of fæces through the intestines or delay in their evacuation

**Etiology** The following varieties are described *Intestinal constipation* There is delay in the passage of the fæces through the intestines usually in the colon *Rectal constipation* or *Dyschezia* Here the intestinal transit time is normal but the fæces accumulate in the pelvic colon and rectum *Greedy colon* Excessive absorption reduces the bulk of the fæces

**Intestinal Constipation** The delay may be due to 1 The small bulk of fæces as would result from insufficient food from a diet poor in residue (such as cellulose) from insufficient fluid intake from excessive fluid loss as in diabetes mellitus or insipidus from excessive sweating or from excessive absorption as in greedy colon

2 A large bulk of fæces, as in heavy eaters causing difficulty in propulsion

3 Weakness of the intestinal wall This may be congenital or acquired due to senile changes or to prolonged wasting diseases

4 Nervous lesions Peristalsis may be disturbed by reflex inhibition as in peritonitis or by emotional central inhibition or the reflex activity may be lowered owing to chronic inflammation of the intestinal mucous membrane or lack of stimulus in the food Irregular contractions causing spastic constipation may result from an irritant in the bowel

5 Obstruction as with new growths of the intestine stercoliths bands adhesions volvulus intussusception and accessory loops (redundant colon)

afternoon a similar state of affairs recurs There is usually no diarrhœa during the night Gastro colic fistula is described on p 47 Hypo thyroidism can be demonstrated by a B M R determination The clinical findings with the other causes of diarrhœa are considered under their respective headings

In all cases of diarrhœa the stools should be examined by naked eye as regards colour appearance presence of mucus blood froth etc and in the laboratory for the presence of undigested food residue the fat and bile content and the presence of pathological organisms

**Treatment** In lienteric diarrhœa all food leaving an irritating residue should be avoided food should not be taken very hot or ice cold A mixture is given containing Tnc belladon m 5 to 10 sod brom gr 5 sp chlorof m 5 aquam ad fl oz ½ Fl oz ½ t d s a c The dose of belladonna is varied according to the result produced In achlorhydria the use of acid hydrochlor dil m 30 to 90 t d s p c in half a glass of water often checks the diarrhœa In hypothyroidism thyroideum gr ½ to 1 daily will often cure the diarrhœa The nocturnal diarrhœa of diabetes is best treated by properly controlling the diabetes by means of diets and insulin and by the injection of a crude liver extract 2 to 4 ml intramuscularly for 6 days followed by a weekly injection of 4 ml In any case of long standing diarrhœa associated with anæmia folic acid therapy is worthy of a trial 50 mg being given daily by mouth for 4 to 6 days In the successful cases reported the response has been rapid In cases of diarrhœa associated with lamblia intestinalis mepacrine mg 100 tab t i d by mouth may cause a speedy improvement It should be given for a week The treatment of the other forms of diarrhœa is considered under the respective headings of the causative diseases

## Visceroptosis

*(Glénard s Disease Splanchnoptosis Enteroptosis Chronic Intestinal Invalidism)*

**Definition** A syndrome characterised by downward displacement of the abdominal viscera with disturbed function of the affected organs and of the mind

**Etiology** Various theories have been propounded to account for the visceral displacement These include A congenital malposition the pressure of corsets chronic peritonitis at the mesenteric root with contraction of newly formed connective tissue a pendulous abdomen with excess of intra abdominal fat a long narrow thorax with wasting and loss of intra abdominal fat weakness of the abdominal and pelvic muscles with lowering of the intra abdominal pressure (this is however normally a negative pressure) intestinal stasis causing dropping of the colon and secondary deficient hepatic function faulty postural habits thoracic breathing and weakness of the transversalis abdominalis muscles X ray examinations with an opaque meal have shown that the stomach may lengthen but it does not drop the upper point of attachment remaining fixed The stomach empties by muscular contractions and not by gravity The lowest point of the greater

by injecting with a syringe 2 to 6 fl oz of warm olive oil into the rectum at night, to be retained during the night

Such laxatives as paraffin liq m 60 to 120 t d s p c, agar gr 60 to 240 t d s, Cascara Evacuant, m 30 to 90 at night Normacol 1 to 2 heaped teaspoonfuls or infusion of senna pods, made by standing the pods in cold water for 24 hours may be used as required The paraffin and cascara may be mixed by adding to the required amount of Cascara Evacuant an ounce of water and floating the paraffin on top In spastic constipation tinc belladon m 10 to 15, or atropin sulph gr 1/200 in m 60 of water may be taken 2 or 3 times a day before food

## Diarrhœa

**Definition** The passage of loose or watery motions

**Etiology** Diarrhœa may be acute or chronic It is a symptom of many diverse conditions and may be due to —

1 *Nervous Causes* These result in the rapid transit of fæces through the intestines *The diarrhœa may be produced by emotion as by an* examination a public speech or a journey in a train in which there is no lavatory accommodation In lienteric diarrhœa there is an accentuation of the gastro colic reflex several loose actions of the bowels occurring after meals Diarrhœa in Graves disease may also be due to nervous excitability Appendicitis may cause a reflex diarrhœa

2 *Gastric Causes* Rapid stomach emptying as in achlorhydria, *gastro colic fistula, and in some cases of gastro jejunostomy*

3 *Local Intestinal Causes* These are numerous and include Irritation of the mucous membrane from foods with much residue food poisoning drugs such as arsenic toxins excreted in the bowel as in septicæmia pneumonia and uræmia catarrh of the mucous membrane as in the pre eruptive stage of measles tuberculosis enteric fever dysentery cholera lambliasis sprue cœliac disease summer *diarrhœa a tumour masses of hard fæces amyloid degeneration of the* intestines and leukæmic infiltration of the intestines Cirrhosis of the liver with portal congestion passive hyperæmia secondary to heart failure and colitis of various types (see p 53)

4 *Mesenteric Causes* Tabes mesenterica and obstruction of mesenteric lacteals causing chylous diarrhœa

5 *Deficiency of Pancreatic or of Biliary Secretion*

6 *Carbohydrate Fermentation* Associated with excessive intake of carbohydrate foods and deficient exercise

7 *Hypothyroidism*

8 *Diabetes Mellitus* Diarrhœa may occur in patients in whom the diabetes is not properly controlled and who suffer from diabetic neuritis It may be an example of a neuropathy affecting the sympathetic nerves of the colon The diarrhœa occurs at night and is intermittent

**Clinical Findings** These must vary with the underlying cause In lienteric diarrhœa the patient is usually of a nervous type a child or an adult a formed motion may be passed after breakfast but during the morning there are several loose motions There is often great urgency so that immediate defæcation cannot *be restrained In the*

**Course and Complications** The course is insidious and progressive and unless adequate treatment is given early the patient gradually develops into a chronic intestinal invalid Complications include Duodenal ileus Dietl s crises and torsion of the pedicle of the spleen

**Prognosis** This is unfavourable as regards complete restoration to health although the condition is not fatal

**Treatment.** *Prophylactic* Children should be trained to adopt a correct bodily poise exercises should be given to strengthen the abdominal muscles and expand the chest Any tendency to constipation should be corrected

*Curative* Rest The treatment should be started with a complete rest in bed for 2 weeks the foot of the bed being elevated 6 to 9 inches on blocks The patient should then gradually get up and about again but only a restricted amount of exercise should be taken Exercises should be initiated during the rest treatment and continued subsequently The costal angle is expanded by inspiratory breathing to elevate the lower ribs and the abdominal muscles strengthened by active contractions with the patient supine An abdominal support such as the Curtis belt may give a feeling of relief when the patient is up but should be discontinued as soon as the abdominal tone increases The patient should always lie down and push the abdominal viscera up with the hand when putting on the support

Diet An ordinary mixed diet is the best If there is much flatulence or gastric distention the meals should be taken dry and excess of carbohydrate avoided as for flatulent dyspepsia (see p 26) but at least 8 pints of fluid should be taken in the 24 hours apart from meals The patient should lie down on the right side for half an hour after meals

Drugs Sedatives such as bromides should be given to calm the nervous system and aid sleep For constipation a mixture of Cascara Evacuant m 30 to 90 to which is added water fl oz 1 and paraffin liq fl oz ½ should be taken at night An attempt should also be made to improve the mental outlook of the patient and her reaction to external stimuli

## Proctalgia Fugax

This is characterised by recurring paroxysms of severe rectal pain occurring chiefly by night It is most common in males between the ages of 30 and 50 The attacks may last for a few minutes up to half an hour The patient may be unable to move in bed during the paroxysm he feels faint becomes pale and perspires The condition is usually considered to be a psychoneurosis No local cause can be found to account for it but it has been suggested that the pain is due to cramp of a portion of the levator ani muscle Attacks occasionally occur by day especially if the patient is overtired

Treatment Relief may be obtained in some cases by eating or by drinking a glass of cold water by a change of position by a hot water bottle applied to the anus or by passing flatus or fæces One sufferer who experienced very severe diurnal attacks obtained relief by taking amphetamine at the onset

curvature of the stomach lies below the umbilicus (inter iliac line) in the erect position in about 80% of healthy people the lesser curvature is below this plane in about 20% of healthy people, and the pylorus is below the transpyloric plane in about 70% of such people The transverse colon may be low without any accompanying symptoms Some of the symptoms may be due to vascular disturbances with low blood pressure and splanchnic dilatation It is evident that displace ment of viscera alone does not produce the symptoms of visceroptosis There must also be disturbance of function The chief physical defects producing the syndrome are a poor muscle tone, postural errors and a disturbance of nutrition, usually wasting There may be an associated neurasthenia *Predisposing causes* 1 Age Usually between 20 and 40 2 Sex Females predominate about 10 to 1 3 Abdominal distention, due to repeated pregnancies but the abdominal wall may be very lax without any associated symptoms 4 Severe illnesses causing wasting

**Pathology** The kidneys spleen, liver and cæcum may be unduly mobile The colon may lie low in the abdomen and redundant loops be present

**Clinical Findings** The patient is usually a young or middle aged woman A history can generally be obtained of progressive ill health The ability to carry out physical or mental work deteriorates interest and concentration flag easily Various abdominal symptoms are present such as constipation intestinal or gastric flatulence discomfort and fulness after meals, a dragging sensation relieved by lying down, pain in the epigastrium around the umbilicus or in either iliac or hypo chondriac region There may also be pain or aching in the lumbar regions or over the sacro-iliac joints and neurasthenic symptoms The patient is often susceptible to cold and the circulation is poor in the hands and feet Rheumatic pains may be felt in various parts of the body

*On Examination* The patient is generally thin, with a tendency to kyphosis in the dorsal region, the chest is long and narrow, and the subcostal angle acute On standing the abdominal wall may be seen to bulge below the navel and the tone of the abdominal muscles is poor When the patient lies the kidneys may be found to be mobile the pulsation of the aorta is easily felt, a stomach splash is in some cases elicited more than 4 hours after the last meal and the contents of the pelvic colon may be palpable The blood pressure is usually low such as 106/68 mm Hg In other cases the patient may be squarely built with a pendulous abdomen A barium meal will reveal the situation of the stomach and intestines the rate of passage of the meal through the alimentary tract and the presence and type of constipation

**Differential Diagnosis** A diagnosis cannot be made until every effort has been taken to exclude other causes for the patient s symptoms These include chronic appendicitis or cholecystitis a gastric or duodenal ulcer abdominal adhesions after operations chronic peritonitis and a gastric or intestinal growth Special examinations include a test meal barium meal and enema cholecystogram and investigation of the fæces for occult blood

cavity Pearly whitish areas of fat necrosis may be seen in the pancreas omentum mesentery and retro peritoneal fat The toxæmia results from autolytic substances produced in the gland

**Clinical Findings** The patient is suddenly taken ill with intense pain in the epigastrium and across the lower part of the back of the chest He feels sick vomits and collapses

*On Examination* In a severe case the patient is desperately ill pale cold sweating and at times cyanosed The epigastrium is extremely tender and there is some but not very marked muscular rigidity in the epigastrium Bluish mottling of the abdominal wall may be noted There may be definite tenderness in the left costo vertebral angle The patient does not keep still as he does with a perforated gastric ulcer The abdomen becomes distended the bowels are confined but flatus is passed No intestinal movements can be heard with the stethoscope The temperature is usually sub normal unless suppuration or gangrene is present The pulse is frequent and feeble The vomit often contains bile The serum amylase is usually well over 200 units per 100 ml serum and the urine may contain sugar and acetone bodies Mild cases also occur in which the diagnosis is established by the diastase test

**Differential Diagnosis** Acute pancreatitis resembles in some respects other acute abdominal emergencies such as a perforated gastric or duodenal ulcer or intestinal obstruction In perforation of a peptic ulcer a serum amylase figure of about 200 may be obtained Coronary disease or acute cholecystitis may be simulated in other cases The characteristic features of acute pancreatitis are the age of the patient the frequent history of gall bladder trouble the absence of a history pointing to a gastric or duodenal ulcer the cyanosis the persistent vomiting collapse and above all the high serum amylase figure

**Course and Complications** If untreated the course is generally rapidly progressive the patient being overwhelmed with toxæmia Suppuration or gangrene may occur as complications or a pancreatic pseudo cyst may form in the lesser peritoneal sac Diabetes mellitus has been recorded developing five years after a successful operation for acute pancreatitis

**Prognosis** This has been improved by modern methods of treatment Sudden death may be due to acute hæmorrhagic pancreatitis When mumps is the cause of the disease the prognosis is good as suppuration and necrosis of the gland do not occur

**Treatment** In mild cases rectal salines containing 5% dextrose and intramuscular injections of pethidine hydrochlor mg 50 to 100 should be given In more severe cases continuous gastric suction through a nasal tube should be applied to prevent the stomach contents entering the duodenum and stimulating pancreatic secretion The blood volume and electrolytic balance must be maintained by con tinuous intravenous drip injections Pro Banthine mg 30 injected intramuscularly every 6 hours may relieve the pain the drug acting as a sympathetic ganglion blocking agent Crystalline penicillin should

## THE PANCREAS

**Introductory** Affections of the pancreas may give rise to very diverse clinical and laboratory findings, according to their nature and to the disturbance of pancreatic function The effects produced may be grouped as follows —

*Disturbances of Internal Secretion* These may be demonstrated by glycosuria, deficiency of insulin results in diabetes mellitus

*Disturbances of External Secretion* Examination of the fæces may show an excess of fat (steatorrhœa) The normal dried fæces contain about 25% of fat and about 5 G of fat are excreted in the fæces in 24 hours With pancreatic insufficiency the fat content may rise to 70% or 80%, the greater part of which is composed of neutral fat The patient must not take any liquid paraffin by mouth for four days before the specimen of fæces is collected Undigested muscle fibres may also be present (azotorrhœa) The urinary diastase normally measures between 6 6 and 30 units, but in some pancreatic lesions such as acute hæmorrhagic pancreatitis it rises to 200 or even 2 000 units Examination of the duodenal contents or of the fæces may show a diminution or absence of trypsin The normal amount of trypsin in 1 G of wet fæces is 900 to 1 500 units

*Pressure Symptoms* A tumour of the head of the pancreas may press on the common bile duct and cause obstructive jaundice or ascites may result from compression of the portal vein

*Nervous Symptoms* Acute pancreatitis may be accompanied by pain in the epigastrium or back vomiting and shock due possibly to irritation of the solar plexus

### Acute Pancreatitis

(*Hæmorrhagic Suppurative and Gangrenous Pancreatitis Pancreatic Necrosis*)

**Etiology** In many cases it is now believed that acute pancreatitis or pancreatic necrosis is due to thrombosis of the pancreatic artery The view that the condition is due to regurgitation of infected bile along the pancreatic duct is losing favour Virus pancreatitis is a blood borne infection which may occur in mumps and occasionally in infective hepatitis and it is possible that in some cases other infections are carried to the pancreas in the blood stream *Predisposing causes* 1 Age Usually over 40 years 2 Sex Males predominate 3 Gall stones and infection of the biliary passages 4 Other diseases Such as mumps enteric fever and small pox 5 A bout of drinking

**Pathology** The steapsinogen and trypsinogen of the pancreatic secretion become activated in the gland This can be effected by bacteria Further trauma or hæmorrhage resulting in a local autolysis of pancreatic tissue produces an activating substance which converts trypsinogen into trypsin The pancreas and adjacent peritoneum show varying degrees of change such as œdema hæmorrhage fat necrosis or gangrene Local suppuration may also occur Fluid containing blood may be present in the lesser peritoneal sac or in the general peritoneal

should be performed The common bile duct may also require surgical drainage Cholecystenterostomy may be required to relieve jaundice

## Fibrocystic Disease of the Pancreas

*(Congenital Pancreatic Deficiency Fibrosis of Pancreas Cystic Fibrosis of Pancreas)*

**Etiology** The cause is still unknown It has been suggested that there is a generalised disorder of mucus secreting glands a mucosis the mucus secreted being scanty and excessively viscid This would mean that in the pancreas the mucus secreting ductal cells are affected with secondary pancreatic changes Other suggestions put forward include a genetically determined malformation of the duct system or primary vitamin A deficiency resulting in obstruction of the pancreatic ducts by desquamated epithelial cells and an increased liability to infection in the lungs Although a serious vitamin A deficiency is usually present it cannot be considered to be the cause of the disease

**Pathology** The exocrine glands are particularly affected the pancreas liver lungs sweat and salivary glands being involved The ducts and acini of the pancreas are dilated and there is atrophy of external secreting tissue The islets of Langerhans are unaffected The lungs show areas of suppurative bronchitis bronchiectasis and terminal bronchopneumonia or abscess Areas of focal biliary fibrosis may be found in the liver Calcium salts may be deposited in the kidneys

**Clinical Findings** The patient is an infant and three types of disease are described The baby may die during the first or second week of life on account of meconium ileus or stenosis or atresia of the intestine Meconium ileus implies intestinal obstruction in the newborn due to meconium which is thick and mucilaginous owing to lack of trypsin during intra uterine life The intestines may rupture and meconium peritonitis result In the second group the symptoms are chiefly pulmonary cough cyanosis clubbing of the fingers with X ray findings of bronchiectasis or cystic spaces in the lungs The third group resembles cœliac disease with large stools intolerance of fats and carbohydrates but in addition there are signs of bronchitis or bronchiectasis The trypsin of the duodenal contents or fæces is reduced or absent and this is the most important of the laboratory tests The fæcal fat is over 30% and is well split The vitamin A absorption curve and the oral dextrose tolerance curve show flat readings Increased sweating may occur with excessive sodium loss in hot weather and there is liability to cramps and heat prostration or even death

**Prognosis** Death often occurs before the age of 14 months from pulmonary infection but I have seen a case in which the diagnosis was first made at the age of 6 years and who responded well to treat ment

**Treatment** If there is bronchiectasis postural drainage should be practised daily and antibiotics such as aureomycin or streptomycin given for 2 weeks Pancreatin gr 30 (six gr 5 tablets) should be given with

be injected intramuscularly in doses of 1 million units every 12 hours In very severe cases or if there is doubt whether the condition is one of perforated peptic ulcer a laparotomy should be performed and drainage of the lesser peritoneal sac effected The gall bladder should also be removed if it appears diseased

## Chronic Pancreatitis

**Etiology** Chronic inflammation of the pancreas is usually due to bacterial infection of the pancreatic lymphatics the organisms being derived from the gall bladder or the intestine It may also be secondary to obstruction of the pancreatic duct as by a gall stone in the ampulla of Vater, a pancreatic calculus inflammation of the pancreatic ducts or carcinoma of the pancreas Arteriosclerosis of the pancreatic vessels syphilis or hæmochromatosis are also causative factors

**Pathology** In chronic interstitial pancreatitis the fibrosis may be interlobular, when it is often localised to the head of the gland, or interacinar when it is more diffuse In chronic catarrhal pancreatitis there is inflammation of the pancreatic ducts

**Clinical Findings** *Chronic interstitial pancreatitis* The patient is usually an adult who complains of epigastric discomfort or pain or of aching in the back flatulence and the passage of bulky, pale and pasty stools *Chronic catarrhal pancreatitis* The patient has periodical attacks of nausea shivering pain in the epigastrium and the left side of the back vomiting and diarrhœa with large motions

*On Examination* Epigastric tenderness is present the conjunctivæ may be slightly icteric the stools contain an excess of fat and undigested muscle fibres In some cases the only symptom is a persistent painless jaundice due to pressure of the head of the pancreas on the common bile duct The urine may contain bile pigment and glucose especially in the case of interacinar pancreatitis and the serum amylase figure may be raised

**Differential Diagnosis** Chronic pancreatitis must be diagnosed from sprue and from digestive disturbances due to biliary insufficiency In sprue the history of residence abroad the characteristic tongue and anæmia and the excess of split fat in the fæces usually serve to establish the diagnosis With biliary insufficiency there is also excess of split fat in the fæces and diminution of bile pigments and bile salts

**Course and Complications** The course extends often over several years the patient gradually losing weight The common bile duct may be compressed in the head of the pancreas with resultant jaundice

**Prognosis** This is unfavourable as regards complete recovery

**Treatment** *A fat poor diet should be given* Eggs should not be eaten The following articles are permitted Toast thin bread and butter, minced meat fish such as cod hake or haddock green vegetables fruit and salads The patient should take no alcohol Pancreatin gr 30 (six gr 5 tablets) may be given t i d after meals Calomel gr ½ should be taken at night and a small dose of salts (mag sulph gr 30) in the morning If there is cholecystitis or gall stones cholecystectomy

medial border of the second part of the duodenum due to pressure by the growth in the head of the pancreas The urine may contain bile pigment and sugar the motions may be pale and contain an excess of split fat

**Differential Diagnosis** It is not generally possible to distinguish a growth in the head of the pancreas from one in the bile ducts The enlarged gall bladder emaciation and progressive jaundice are indicative of growth as opposed to chronic pancreatitis A barium meal and fractional test meal serve to exclude carcinoma of the stomach

**Course and Complications** The course is rapidly progressive The growth may erode into the stomach and cause hæmatemesis Secondary deposits may occur in the long bones with a resultant anæmia This is usually hypochromic but at times megalocytic, when nucleated red cells and less often megaloblasts are present The abdominal and mediastinal glands may also be invaded and ascites may develop

**Prognosis** Death usually occurs in the course of a few months

**Treatment** Cholecystenterostomy may be performed to relieve the jaundice and itching but there is no curative treatment

## Cysts of the Pancreas

**Pathology** The following varieties of cysts may occur 1 The retention cyst This has an epithelial lining and forms as the result of obstruction of the pancreatic ducts as by a stone or chronic pancreatitis 2 The pseudocyst This is formed by the lesser peritoneal sac and results from trauma or acute pancreatitis 3 The proliferative cyst and cyst adenoma These are multilocular and often found near the tail of the pancreas They may form part of congenital polycystic disease of the kidneys and liver 4 The hydatid cyst (see p 749) and dermoid cyst

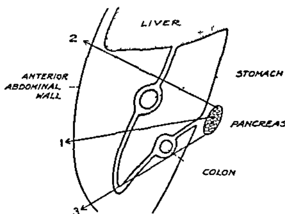



FIG 6 DIAGRAM SHOWING POSITIONS IN WHICH A PANCREATIC CYST MAY PRESENT

**Clinical Findings** The patient is usually over the age of 20 There may be a history of abdominal trauma There may be no symptoms or the patient may complain of pain in the epigastrium or between the shoulders and of nausea vomiting constipation jaundice and swelling of the abdomen and legs

*On Examination* The cyst may be felt as a rounded fluctuating swelling in the epigastrium It commonly presents between the stomach and transverse colon but may show above the stomach or below the transverse colon as indicated in the diagram (see Fig 6) Its position

each meal The diet should be high in protein and low in fat In addition halibut liver oil should be given by mouth m 5 to 10 twice daily, and vitamin A 100 000 units, injected intramuscularly every 3 months

### Tumours of the Pancreas

Simple tumours and gumma are rare, an adenoma of the islets of Langerhans may give rise to *hyperinsulinism,* with symptoms of spontaneous hypoglycæmia such as faintness, sweating headache etc (see p 660) Cardiac symptoms predominate in some cases either *anginal in type or resembling the effort syndrome* Neuropsychiatric symptoms may predominate such as episodes of uninhibited emotional behaviour cured by removal of an islet tumour Further people with cerebral dysrhythmia as shown by the electro encephalogram, are very susceptible to falls in the blood sugar level The dextrose tolerance curve after 3 days on a diet containing 300 G carbohydrate shows a low fasting value of about 70 mg /100 ml a rise to about 120 mg after 50 G of dextrose by mouth and subsequently low readings at 2 3 and 4 hours A hypoglycæmic attack may occur during the test Spontaneous hypoglycæmia may also be due to functional hyperinsulinism and hepatic disease In functional hyperinsulinism the fasting blood sugar is usually normal in hepatic disease the fasting blood sugar is low, but the curve shows a high plateau similar to that in diabetes mellitus In the latter there is no hyperinsulinism but faulty *glycogenesis in the liver It may occur in such conditions as cholan* gitis toxic hepatitis Von Gierke s disease (see p 104), diffuse car cinomatosis and fatty degeneration of the liver Malignant tumours are usually carcinomata sarcomata are rare Hyperinsulinism was first described in 1927 in association with a carcinoma arising in the islets of the pancreas

### Carcinoma of the Pancreas

**Pathology** The carcinoma is usually primary and situated at the head of the gland Secondary deposits in the pancreas are less common the primary growth being usually in the stomach The gall bladder may be enlarged owing to compression of the common bile duct by the growth or by secondary glands in the portal fissure

**Clinical Findings** The patient is usually a male over the age of 40 *He complains of general weakness* loss of appetite pain in the epi gastrium passing through to the back, progressive jaundice and itching of the skin *In some rare cases the symptoms are those of recurrent* hypoglycæmia in others there are no signs the patient complaining only of severe pain in the back in the lower dorsal region *or of girdle* pains, which are relieved by heat and by sitting bent forward

*On Examination* The patient is wasted and the skin is jaundiced to a variable degree sometimes only slightly but it may be dark green (' black jaundice ) The gall bladder may be palpable but the pancreatic tumour can rarely be felt unless it is situated in the body or tail of the gland The barium meal may show indentations on the

bilirubin such as carotene or mepacrine The normal value in the blood is 2 to 8 units In *latent jaundice* there is an excess of bilirubin in the blood usually between 1 to 3 mg /100 ml but no jaundice is found clinically The direct and ' indirect and total serum bilirubin are determined When the jaundice is mainly due to increase in direct ' pigment the condition may be obstructive or toxic and infective Latent jaundice is met with in infective hepatitis cirrhosis hepatitis and the hæmolytic anæmias In *dissociated jaundice* there is an excess of bile salts in the blood without a corresponding rise in the bilirubin content This may cause pruritus and bradycardia in certain cases of cirrhosis hepatis Bile pigments in the urine can be tested for clinically With a deficiency of bile in the intestine the stools become fatty there being an excess of split fat

2 *The metabolic function* In *the galactose tolerance test* 40 G of galactose are given by mouth and the blood galactose curve determined and the urine examined for galactose The blood samples are examined every 30 minutes for 2 hours Normally the blood galactose does not rise above 10 to 40 mg /100 ml The sum of the four blood galactose readings called the galactose index is normally about 70 mg An index over 160 indicates hepatocellular damage Normally the urinary excretion of galactose in 5 hours is less than 3 G A figure over 4 G is suspicious and one of over 6 G indicates severe liver damage

3 *The hæmopoietic functions* *The alkaline plasma phosphatase* rises above the normal of 7 to 13 King Armstrong units/100 ml in some cases of obstructive jaundice It has been claimed that readings above 50 units signify obstructive jaundice above 20 units indicate non obstructive jaundice and normal figures are found in hepatic jaundice It must be remembered that the alkaline phosphatase is raised in such conditions as Paget s disease rickets hyperparathyroidism osteo malacia osteogenic sarcoma and other primary skeletal tumours *The plasma proteins* may also be affected in liver disorders the plasma albumin falling below the normal low level of 3 8 G /100 ml There are many other causes for a fall in the plasma albumin *The Takata Ara test* depends upon the precipitation of serum by varying strengths of perchloride of mercury solution It is likely to be positive in cirrhosis and negative in carcinoma of the liver The test is usually positive in infective hepatitis the strength of the reaction varying with the degree of liver damage Other flocculation tests include the colloidal gold and the thymol turbidity The normal colloidal gold figure is 0 to 1 unit and the normal thymol turbidity reading is 0 to 4 units The flocculation tests are usually positive in infective hepatitis and negative in obstructive jaundice

4 *The excretion of foreign substances* *The bromsulphthalein test* consists in the intravenous injection of 5 mg /kg body weight of bromsulphthalein and removal of a specimen of blood at 5 minutes and 45 minutes The dye content of the sera is estimated Normally the 5 minute specimen contains between 15 and 85% of the dye and the 45 minute specimen between 0 and 5% A figure above 20% in the second specimen indicates liver damage

can usually be identified with the help of a barium meal or enema The cyst fluid removed by aspiration may contain pancreatic ferments

**Differential Diagnosis** The pancreatic cyst may be mistaken for a hydatid cyst of the liver, a mesenteric cyst, a hydronephrosis or rarely for an ovarian tumour The anatomical relations usually suggest the correct diagnosis

**Course and Complications** There is usually gradual enlargement of the cyst but internal hæmorrhage causes rapid distention Rupture *results in local or general peritonitis* Malignant degeneration may occur

**Prognosis** A complete cure, even after operation, is improbable as recurrence often takes place Post-operative diabetes mellitus has also been recorded

**Treatment** An exploratory laparotomy usually permits the cyst to be drained, complete removal being generally impossible

### Pancreatic Calculi

**Etiology** Calculi form as the result of obstruction of or infections in the pancreatic ducts

**Pathology** The calculus may be composed of a mixture of calcium carbonate and phosphate, or of calcium oxalate, carbonate and cholesterin Calculi are usually multiple not facetted and vary in size from a small granule to an inch in diameter The pancreas shows chronic inflammatory changes and gall stones are often present

**Clinical Findings** The patient is usually a male over the age of 40 There may be no symptoms or attacks of epigastric colic and vomiting occur *the pain radiating to the region of the left scapula*

*On Examination* The patient may be tender in the epigastrium and also jaundiced The fæces show azotorrhœa or steatorrhœa and the greyish white calculi may be passed in the stools If the calculi are rich in lime, they are shown by X ray examination

**Differential Diagnosis** The colic resembles biliary colic but the pain tends to radiate to the left

**Course and Complications** Repeated attacks of colic may occur Complications include diabetes mellitus pancreatic abscess and peritonitis An abscess may discharge through the abdominal wall

**Treatment** Sedatives such as morphin sulph gr ¼ or inhalations of chloroform are *required as for biliary colic In some cases the* calculi have been removed by operation

## THE LIVER, GALL-BLADDER AND BILE DUCTS

**Introductory** In studying diseases of the liver certain special methods of investigating hepatic function are available

The hepatic efficiency tests of most clinical value include 1 *The investigation of the pigmentary function* This involves tests for the presence of bile in the blood urine and fæces Bile pigments in the blood are shown by the van den Bergh test The normal amount of plasma bilirubin is 0 2 to 0 8 mg /100 ml The icterus index is a less accurate test as the serum may be coloured by other substances than

**Passive Hyperæmia** This usually results from back pressure due to heart failure There is congestion and anoxia of the central zone of the liver lobules producing the 'nutmeg' liver

**Clinical Findings** The patient may complain of pain in the liver region

*On Examination* In addition to the evidence of cardiac disease the liver is enlarged and expansile pulsation may be detected on bimanual palpation especially if tricuspid regurgitation is present There may also be jaundice

**Treatment** The underlying cardiac lesion must be treated In addition venesection helps to relieve the congestion

### Infarction

The intralobular branches of the portal vein may be blocked by an embolus or by thrombosis An embolus may also occur in the hepatic artery Thrombosis may develop in the larger branches of the portal or hepatic veins The embolus may consist of blood clot or of a portion of new growth The infarct is hæmorrhagic unless due to obstruction of the hepatic artery when it is anæmic It is generally deep seated and gives rise to no symptoms during life unless there is pain from an associated perihepatitis

### Perihepatitis

**Definition** Inflammation of the capsule of the liver This may be acute or chronic

### Acute Perihepatitis

**Etiology** This may be secondary to a liver abscess gumma hydatid cyst cholangitis etc or it may form part of a general or local acute peritonitis

**Clinical Findings** The patient complains of pain in the region of the liver near the angle of the right scapula or at the tip of the right shoulder

*On Examination* There is diminished movement on the affected side of the chest the liver may be tender on palpation and a localised friction rub can be felt or heard

**Treatment** The pain is best relieved by immobilising the side of the chest by strapping

### Chronic Perihepatitis

This may be local or diffuse

**Local Perihepatitis** This may be due to local affections of the liver such as a gumma or cyst or occur in association with passive hyperæmia due to heart disease or adjacent to an inflamed gall bladder It may also form part of a tuberculous or malignant peritonitis

**Clinical Findings** There are usually no symptoms but localised pain may occur

**Diffuse Perihepatitis (Sugar iced Liver)** This is associated with chronic proliferative peritonitis (see p 119) A thick white fibrous

5 *The prothrombin response to vitamin K* This is of value in surgical jaundice The prothrombin in the blood is estimated If it is low mg 100 of vitamin K (Synkavit mg 100 in 2 ml) are injected intramuscularly The prothrombin is estimated again after 24 to 48 hours If the value is now normal it indicates biliary obstruction If there is no response there is probably gross liver damage

6 *Liver puncture biopsy* A small specimen of liver tissue is removed by liver puncture, using a special trocar and cannula and examined histologically

7 *X ray examination* Cholecystography and cholangiography reveal the size shape filling capacity and contractility of the gall bladder and the presence of calculi and the condition of the bile ducts In some cases calculi are seen in a direct radiogram

## THE LIVER

### Hepatitis

Infective hepatitis is the most common cause of acute inflammation of the liver Less often it is associated with hepatic abscess secondary syphilis cloudy swelling acute yellow atrophy of the liver and toxins and infections affecting the liver Cirrhosis of the liver may be regarded as a variety of chronic hepatitis

### Malformations and Displacements

The malformation which is of the greatest importance clinically is the Riedel s lobe This is a tongue like downward projection from the right lobe of the liver, which may be mistaken for an enlarged gall bladder or for the right kidney It usually occurs in women

The liver may be displaced downwards as by a subphrenic abscess, or upwards by an abdominal tumour or ascites Hepatoptosis is a term applied to prolapse of the liver It is more common in women and may give rise to abdominal dragging to pain or to biliary colic

*On Examination* An abdominal tumour is felt which can be pushed back into the space normally occupied by the liver

### Hyperæmia

**Active Hyperæmia** This may be associated with a chill, especially *in tropical countries in individuals who have suffered from malaria* or dysentery It may also result from over eating associated with a sedentary life and chronic constipation It is often known as a " chill on the liver "

**Clinical Findings** The patient complains of headache nausea constipation and a sense of fullness or pain in the region of the liver

*On Examination* The tongue is furred and the liver may be just palpable and somewhat tender

**Treatment** The patient should be kept in bed for a few days on a milk diet, and aperients given such as calomel gr 2 nocte with mag sulph gr 60 to 120 mane Hot applications to the liver area are comforting Subsequently a belt should be worn over the upper abdomen

usually a leucocytosis but the systemic blood culture is often sterile The temperature is irregular it may be remittent or intermittent

**Differential Diagnosis** The increase in the gravity of the patient's condition with the rigors and hectic temperature suggest pyæmia and the evidence of liver involvement renders the diagnosis of portal pyæmia clear In many cases however it is difficult, if not impossible, to diagnose during life There may be no definite localising signs pointing to the primary source of the infection

**Course and Complications** The course is progressive Complications such as empyema or a lung abscess may occur

**Prognosis** Death usually occurs in one to six weeks

**Treatment** It is doubtful whether there is any curative treatment available in established cases Treatment is therefore palliative for relief of pain and discomfort

### Thrombosis of the Hepatic Veins

Primary thrombosis of the hepatic veins is known as *Chiari s disease* The symptoms include sharp right upper abdominal pain radiating to the back and in some cases shock nausea vomiting and collapse The liver is enlarged and tender the spleen is enlarged and there is ascites There is no venous engorgement in the neck as is met with in right sided cardiac failure Death may occur from hæmatemesis Secondary hepatic vein thrombosis may be associated with liver disease with septicæmia erythræmia vera or with thrombo phlebitis migrans

### Portal Thrombosis

(*Pylethrombosis Pylephlebitis Adhæsiva*)

**Definition** Thrombosis of the portal vein

**Etiology** Portal thrombosis is most often associated with cirrhosis of the liver or with new growths in the pancreas stomach or liver In other cases it is due to syphilis of the portal vein trauma or to infections in the portal area such as the intestines appendix spleen pancreas or gall bladder It may occur in erythræmia vera and in splenic anæmia

**Pathology** The wall of the portal vein or its branches is generally thickened and the blood is clotted The spleen is usually enlarged but the liver may be normal in size The mesenteric veins alone may be thrombosed with secondary gangrene of the intestine usually in the jejunum

**Clinical Findings** There may be hæmatemesis acute abdominal pain or swelling of the abdomen due to ascites In cirrhosis of the liver hepatic vein thrombosis may precipitate hepatic coma With a mesenteric thrombosis (see p 122) the symptoms resemble those of an acute abdominal emergency

*On Examination* The spleen may be enlarged and dilated veins seen around the umbilicus

**Differential Diagnosis** It is very difficult to make a certain diagnosis in cases of portal thrombosis especially as it is so often associated

coat forms over the liver, which can be stripped off There is often thickening of the omentum ascites, chronic pericarditis, arteriosclerosis and chronic nephritis (Pick s disease) There are usually no toxæmic symptoms and no jaundice

**Treatment** The ascites can be relieved by aspiration and pot iod should be given by mouth in doses of gr 5 to 10 t i d

## Liver Abscess

**Definition** Suppuration in the liver

**Etiology** The abscess may be due to the following causes 1 *Amœbiasis* (*see* p 722) It is then usually single 2 Portal pyæmia The infection may arise in the appendix ("appendicular liver") or in other sites such as the prostate or rectum It may be secondary to suppurative pylephlebitis (see below) The abscesses are usually multiple 3 Arterial pyæmia This is often secondary to otitis media the infection being carried to the liver by the hepatic artery 4 Suppurative cholangitis The infection spreads up the bile ducts 5 Trauma An abscess may form secondary to a wound of the liver 6 Retrograde infection by the hepatic veins This rarely occurs 7 Direct spread of infection This may be due to a subphrenic abscess or empyema of the gall bladder 8 Suppuration in a hydatid cyst 9 Actinomycosis

**Clinical Findings** The clinical picture in amœbiasis and hydatid infection is considered separately (see pp 723 749) With pyæmic abscesses the patient is very ill with a swinging temperature and rigors The liver is usually enlarged and tender, and there may be icterus

**Prognosis** With multiple abscesses this is usually hopeless

**Treatment** No special treatment is available

## Suppurative Pylephlebitis

**Definition** This term is synonymous with portal pyæmia Suppuration occurs in the tributaries of the portal vein in the vein itself and its branches

**Etiology** Suppurative pylephlebitis is most commonly due to an appendix abscess Less often the condition is secondary to an amœbic liver abscess to pus in the gall bladder or bile ducts to infection after rectal operations or pelvic operations in women

**Pathology** The liver is enlarged perihepatitis is usually present, and abscesses may be seen on the surface or only on section of the organ The portal vein or its branches contain disintegrating blood clot with a variable amount of pus

**Clinical Findings** The disease usually begins during convalescence from an operation for an appendix abscess The patient becomes gravely ill with rigors sweating and increase of temperature and pulse rate This is followed in a few days by pain in the region of the liver

*On Examination* The patient looks ill the tongue is dry and furred and there may be slight icterus The liver is found to be enlarged and tender The spleen is not usually enlarged The blood There is

posteriorly The diagnosis is usually suggested by the history of the case the negative Wassermann and the positive blood and intradermal tests

**Course and Complications** The cyst may cure itself by inspissation On the other hand it may continue to grow, it may suppurate or a hæmorrhage may occur into it Rupture may take place into the abdomen stomach intestine etc

**Prognosis** The mortality rate is about 15%

**Treatment.** The cyst should not be tapped If causing symptoms, it should be removed as completely as possible by an open operation Aspiration is dangerous as severe shock or death may occur if any of the fluid enters the peritoneum

### Calcification of the Liver

X ray examination of the upper abdomen may reveal extensive areas of calcification in the liver This is most often due to hydatid infection but it may result from syphilis carcinoma infective or vascular lesions

### Cirrhosis of the Liver

**Definition** Hardening of the liver due to the formation of fibrous tissue The varieties of cirrhosis may be classified as follows 1 Portal 2 Post necrotic 3 Biliary 4 Pigmentary 5 Parasitic 6 Syphilitic 7 Congestive

### Portal Cirrhosis

(*Diffuse Hepatic Fibrosis Alcoholic Cirrhosis Laennec s Atrophic Cirrhosis Hob nail Liver Gin drinker s Liver*)

**Etiology** Clinical experience shows that in the majority of cases cirrhosis of the liver is associated with persistent excessive intake of alcohol The question whether alcohol exerts a direct toxic effect on the liver is not finally decided although there is no doubt that after an attack of infective hepatitis it acts as a liver poison Experimental work by liver puncture in man and by feeding experiments on animals has thrown further light on the etiology of cirrhosis in certain cases Cirrhosis may follow an attack of infective hepatitis A high fat diet fed to animals may cause first fatty changes and later cirrhosis of the liver Further animals fed on a low protein diet may develop hepatic necrosis the deficiency which leads to such changes being sulphur containing amino acids such as methionine and cysteine Cirrhosis may at times develop in man either directly from protein nutritional deficiency or indirectly because excessive consumption of alcohol results in a lowered protein intake or causes a gastro-enteritis which interferes with the digestion and absorption of protein Cirrhosis may also occur in Wilson s disease or in association with ulcerative colitis typhoid fever malaria or hyperthyroidism *Predisposing causes* 1 Age Usually over 40 but cirrhosis may occur in children 2 Sex Males predominate 3 Occupation Especially those working in the liquor trade and commercial travellers 4 Climate and locality It is more common in temperate zones

with other severe diseases The sudden onset of abdominal pain hæmatemesis or ascites is very suggestive

**Course and Complications** *The thrombosis may* persist for many years or rapidly prove fatal Recurrent attacks of hæmatemesis may occur

**Prognosis** This is very *grave*, but *in some cases the patient* survives the attack for several years

**Treatment** If the patient survives the immediate shock anti coagulant treatment should be given (see p 186) and a course of penicillin injections

### Cysts of the Liver

The following varieties of cyst may occur 1 Blood and degeneration 2 Dermoid 3 Lymphatic 4 Endothelial 5 Due to bile duct obstruction 6 Cystadenoma 7 Hydatid 8 Polycystic This forms part of congenital polycystic disease of the kidneys (see p 499) The solitary non parasitic cyst is probably a cystadenoma arising from a congenital aberrant bile duct

**Clinical Findings** Unless the cyst is large it cannot be felt In polycystic disease, however, the enlarged and irregular surface of the liver can often be detected Cysts may rupture into the peritoneum causing severe shock or a fatal hæmorrhage may take place into the cyst A cyst may press on the bile ducts or duodenum suppuration may occur, or the pedicle become twisted

### Hydatid Cysts

**Etiology** The disease is due to infection with the Echinococcus granulosus (T echinococcus) (see p 749)

**Pathology** The cysts may be multiple or single deeply embedded in the liver substance or projecting from the surface The liver is the organ most commonly affected in hydatid disease The structure and contents of the cyst and the changes they may undergo are described on pp 749 750

**Clinical Findings** If the cyst is small or deep seated it may cause *no symptoms* *In other cases the patient complains of aching* or of pain in the region of the liver, and he may give a history of residence *in an infected area*

*On Examination* The liver may be enlarged and a rounded swelling felt depending upon the position of the cyst A hydatid thrill (see p 749) is rarely palpable The blood may show an eosinophilia The Casoni intradermal test consists in the intradermal injection of 0 2 ml of filtered hydatid fluid obtained from a cyst of a sheep A positive reaction is shown by the formation of an urticarial wheal in about 10 minutes followed in a few hours by erythema and œdema around the site of the injection There may also be a febrile reaction

**Differential Diagnosis** A hydatid cyst if palpable, must be distinguished from a gumma or *carcinoma of the liver or an enlarged* gall bladder It may also simulate a pleural effusion if situated

**Pathology** In the early stages the liver is enlarged and fatty it shrinks (Laënnec's atrophic cirrhosis) The surface is irregula projections being called ' hob nails ' These are formed by hyper of liver cells, and are yellowish on section (*Κιρρος* = yellow) The is tough on section owing to the bands of fibrous tissue which sur several lobules In many cases the liver is enlarged with a granular surface and the strands of fibrous tissue are perilobu distribution In other instances while portions of the liver are he in some areas the liver lobules are replaced by newly regenerated cells The spleen may be enlarged A compensatory venous circul is established on account of the portal obstruction The main cha opened up are Veins running from the bare area of the liver t phrenic and intercostal veins *An anastomosis between the œsoph* veins and the coronary veins of the stomach Dilated veins are present in the lower part of the *œsophagus* and in the stomach may be erosions of the mucous membrane or dilated veins Enlarge *of the para umbilical vein of Sappey running in the falciform liga* and connecting the epigastric and portal veins Dilatation of the *around the umbilicus gives rise to the appearance known as the Medusæ* An anastomosis between the inferior mesenteric and *hæmorrhoidal veins Hæmorrhoids are not common* Enlargeme the retroperitoneal veins of Retzius establishing communic *between the portal tributaries and those of the inferior vena cava* Fig 7)

**Clinical Findings** *The patient is usually an adult male ove* age of 40 who gives a history of over indulgence in alcohol for se *years In the early stages the symptoms are due to chronic gast* The patient complains of flatulence, nausea retching or vomiting s *quantities of fluid on rising and of a lack of desire for breakfast* He have headache recurrent attacks of epistaxis and constipation *first symptom in some cases is a hæmatemesis of half a pint or more* the later stages the patient complains of increasing weakness, dysp and perhaps of swelling of the abdomen and legs In other cases t is a history of one or more attacks of jaundice probably due to infe *hepatitis*

*On Examination* The facies is usually suggestive the patient b *well nourished with dilated venules on the cheeks* and the eyes congested and watery The tongue is furred and the breath is o *offensive The liver may be felt enlarged* a little irregular and and the spleen may be just palpable

*Later, the patient presents the typical hepatic facies* the fa thin and the nose prominent the complexion is rather sallow, and dil *venules are seen* on the cheeks and nose The chest arms and are wasted, but there may be swelling of the legs due to œdema *œdema* may be due to various causes such as partial obstructio the inferior vena cava or abdominal lymphatics to loss of blood p tein into the ascitic fluid or to a toxic effect on the capillaries

FIG 7 COMMUNICATIONS BETWEEN THE PORTAL AND SYSTEMIC VENOUS SYSTEMS

A Between the retro peritoneal veins which open into the lumbar and azygos veins and the veins of the intestine especially in those regions where the gut is bound down to the abdominal parietes *e g* the duodenum and ascending and descending colon This anastomosis was described by Retzius

B Veins in the liver substance and capsule may communicate with the phrenic and intercostal veins where the liver and diaphragm are uncovered by peritoneum

C Very rarely the ductus venosus of the fœtus remains patent and connects the left branch of the portal vein with the inferior vena cava

D 1 The falciform ligament contains veins which establish communication between the left branch of the portal vein and the veins of the abdominal wall These vessels are known as the para umbilical veins of Sappey and lie in close relation to the obliterated umbilical vein of the fœtus (ligamentum teres) In portal obstruction a large vein is often found in the free margin of the falciform ligament Rokitansky and Bamberger originally stated that this vessel was the patent umbilical vein Sappey however asserted that the vein was always a para umbilical vessel

D 2 Veins running anteriorly in the falciform ligament connecting the umbilical or para umbilical veins with a superior epigastric vein have been seen post mortem in cases of portal obstruction

E The œsophageal branches of the left gastric vein anastomose with the systemic œsophageal veins which flow into the azygos system

F The gastric vein communicates with the phrenic vein

G Veins in the capsule of the kidney and the perirenal fat communicate on the left side with the veins of the descending colon and on the right with veins of the ascending colon and duodenum Congestion of the renal vessels has been described in cirrhosis and this may account for hæmaturia

H The veins of the descending colon may communicate with the spermatic plexus Varicocele may occur in cirrhosis

I The superior hæmorrhoidal vein a tributary of the inferior mesenteric vein anastomoses with the middle and inferior hæmorrhoidal veins of the systemic circulation

J A communication between the left renal vein and the splenic vein has been described

K A communication between the splenic and azygos veins has been observed

Peritoneal adhesions to the liver stomach duodenum and spleen favour the formation of anastomoses between portal and systemic veins

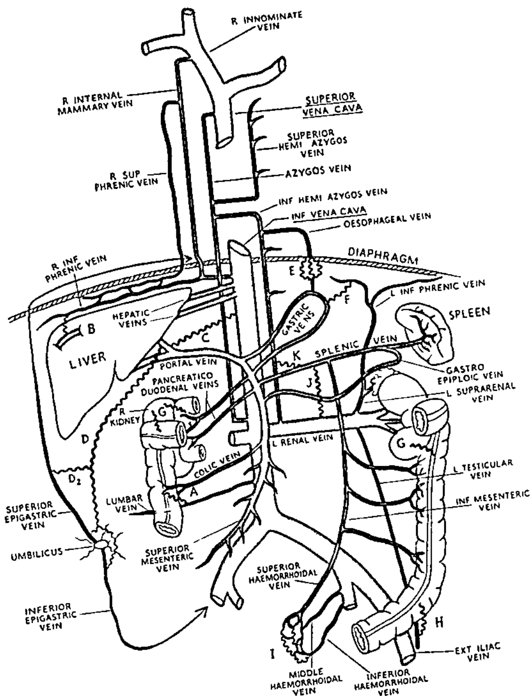

R INNOMINATE VEIN
R INTERNAL MAMMARY VEIN
SUPERIOR VENA CAVA
SUPERIOR HEMI AZYGOS VEIN
R SUP PHRENIC VEIN
AZYGOS VEIN
INF HEMI AZYGOS VEIN
INF VENA CAVA
OESOPHAGEAL VEIN
DIAPHRAGM
R INF PHRENIC VEIN
E
F
L INF PHRENIC VEIN
HEPATIC VEINS
GASTRIC VEINS
SPLEEN
B
C
LIVER
K
SPLENIC VEIN
PORTAL VEIN
GASTRO EPIPLOIC VEIN
PANCREATICO DUODENAL VEINS
J
L SUPRARENAL VEIN
R KIDNEY
G
D
L RENAL VEIN
G
COLIC VEIN
L TESTICULAR VEIN
D2
LUMBAR VEIN
A
SUPERIOR EPIGASTRIC VEIN
INF MESENTERIC VEIN
UMBILICUS
SUPERIOR MESENTERIC VEIN
SUPERIOR HAEMORRHOIDAL VEIN
INFERIOR EPIGASTRIC VEIN
H
I
EXT ILIAC VEIN
MIDDLE HAEMORRHOIDAL VEIN
INFERIOR HAEMORRHOIDAL VEIN
= PORTAL CIRCULATION
= SYSTEMIC VEINS

Fig 7

[T f cp 8

glands are usually found elsewhere The alcoholic history and appearance of the patient are also of value

In the ascitic stages other causes of ascites must be considered (see p 120) Cholæmia or hepatic toxæmia resembles uræmia in some respects clinically and the non protein nitrogen content of the blood may be raised

**Course and Complications** In the early stages the disease may pursue a very slow course and arrest may even occur Complications include Hæmatemesis portal thrombosis pulmonary tuberculosis tuberculous peritonitis hæmorrhagic pleural effusion pneumonia and peripheral neuritis

**Prognosis** This is very grave except in the early stages and the patient usually dies a few years after the diagnosis is made Hepatic coma and hæmatemesis are serious complications

**Treatment** In the early stages the patient must give up all alcohol and spiced foods and no tinctures or spirits must be given as medicines The diet should be rich in meat fish eggs milk fruit green vegetables and carbohydrate containing foods It should contain about 300 G carbohydrate 180 G protein and 100 G fat Brewers yeast ½ oz may be given in milk twice daily or two Beplex capsules a day and aneurin hydrochlor mg 5 taken by mouth daily An intramuscular injection of 5 ml of a crude liver preparation should be given twice a week If there is severe polyneuritis or mental confusion 100 mg of aneurin hydrochlor and 300 mg of nicotinamide should be injected intramuscularly daily The bowels should be kept acting daily with salines such as mag sulph gr 60 mane If the Wasserman reaction is positive a course of pot iod gr 5 to 60 t i d should be given for 2 to 3 months If there is hæmatemesis no morphine must be given, as owing to failure of hepatic detoxication even gr ⅙ may prove fatal

*Ascites* In acute ascites due to alcoholic cirrhosis rest in bed with a mixed diet as described above and no alcohol may cause a rapid disappearance of the fluid In the more chronic types it may be possible to abolish the ascites by a high protein low sodium diet combined with mercurial diuretics and ammonium chloride. After a preliminary paracentesis the patient is given a diet containing 120 G protein with a sodium content up to mg 500 a day By the use of Casilan 60 G in a pint of water during the day it is possible to obtain enough protein without increasing the sodium intake The caloric value of the diet should be about 2 500 The fluid intake need not be restricted Two ml of mersalyl are injected intramuscularly twice a week and ammonium chloride is given by mouth 1 G t i d The ammonium chloride can be given as 0 5 G enteric coated tablets It is also advisable to give potassium chloride 1 G daily After several months it is usually possible to increase the sodium intake without recurrence of ascites A careful watch must be kept for early signs of hepatic coma such as mental changes when the administration of ammonium chloride must be discontinued

Various operations have been devised either to drain away the fluid or to relieve portal hypertension The Talma Morison operation is the

firm and irregular In other cases the liver is not palpable until the fluid has been removed from the abdomen or the liver may have shrunk and not be palpable "*Vascular spiders*" *may be seen on the* face arms, hands and upper part of the trunk They are red and are composed of a central arteriole which pulsates with radiating legs Pressure on the central arteriole with a pin causes the legs and the whole red area to blanch The palms of the hands especially the thenar and hypothenar eminences and the tips of the fingers are sometimes hot and red the so called ' liver palms " Endocrine disturbance may be shown by testicular atrophy and gynæcomasia in males and by hirsuties or disturbance of the periods in females The dilated veins may be seen in the abdominal wall as described above (see Fig 7) A venous hum may be heard over the lower part of the sternum and the liver Symptoms of toxæmia may be present the patient complains of headache inability to concentrate, and drowsiness The skin is usually sallow or slightly icteric and muscular tremors or twitching may be seen This may pass on to hepatic coma The urine is con centrated and may contain bile The blood shows an excess of bile pigment and in the later stages of the disease a macrocytic anæmia may develop Further the plasma proteins are low, or if the total protein content is normal, the globulin figure is higher than the albumin Liver function tests show a moderate rise in the serum bilirubin an increase in the urobilinogen in the urine and the bromsulphthalein test is positive there being an increased retention of the dye

A barium swallow or œsophagoscopy will reveal the presence of œsophageal varices The temperature is often irregular and slightly raised

**Differential Diagnosis** The symptoms in the early stages are those of chronic gastritis or of tenderness over the liver and there may be no indication that the liver is cirrhotic until pressure manifestations occur such as hæmatemesis or until the liver is definitely enlarged Other causes of hæmatemesis such as gastric ulcer must then be excluded The barium meal will afford evidence of gastric ulceration

Enlargement of the liver necessitates a consideration of other causes than cirrhosis such as malignant disease syphilis splenic anæmia, passive hyperæmia amyloid degeneration a hydatid cyst an abscess *leukæmia or Hodgkin s disease In malignant* disease the liver is usually more irregular definite nodules with central umbilication are palpable in some cases and jaundice tends to be more intense In some cases however the liver is infiltrated with secondary deposits and no jaundice is seen The growth is usually secondary to a lesion elsewhere In syphilis the Wassermann reaction is positive and there is ameliora tion of the condition with the administration of iodides In splenic anæmia the spleen is usually considerably enlarged before the liver enlarges In passive hyperæmia there is evidence of heart failure and expansile pulsation may be detected in the liver In amyloid degenera tion the patient is usually suffering from a definite wasting or suppura tive disease The blood examination is of value in the diagnosis of hydatid disease *abscess and leukæmia In Hodgkin s disease enlarged*

**Etiology** The cause is not known Alcohol is probably not a causative factor *Predisposing causes* 1 Age 20 to 30 years but it may occur in children 2 Sex It is more common in women

**Pathology** Post mortem the liver is enlarged with a smooth surface and dark green colour It is firm on section and a fine peri lobular fibrosis is present The larger bile ducts are normal the smaller ones show cholangitis or pericholangitis The spleen is normal or enlarged The portal lymph glands may be enlarged

**Clinical Findings** The disease is very rare The onset is gradual the patient complaining of loss of appetite attacks of nausea or diarrhœa jaundice and itching of the skin Pain may be felt in the region of the liver or of the spleen

*On Examination* The patient is jaundiced but not usually very deeply The liver is found to be enlarged and may extend from the fourth rib to 4 inches below the costal margin It feels smooth and firm The spleen may be palpable There is no ascites The fingers are often clubbed and hypertrophic osteo-arthropathy may be seen in the wrists or other joints The blood There is a hæmolytic anæmia but the leucocytes are often increased up to 12 000 per c mm or more The van den Bergh reaction is indirect The urine This contains urobilin and increased urobilinogen The fæces Stercobilin is diminished

**Differential Diagnosis** It may be confused with acholuric jaundice Banti s disease portal cirrhosis infective hepatitis and xanthomatous biliary cirrhosis The characteristic features are the persistent jaundice of a rather mild degree the absence of ascites and the enlarged liver The red cells show no increased fragility as they do in acholuric jaundice In Banti s disease there is usually a leucopenia and hæmorrhages occur before the onset of jaundice Portal cirrhosis is characterised by a milder degree of jaundice and the tendency to ascites In obstructive jaundice the fæces contain little or no bile In xanthomatous biliary cirrhosis the blood cholesterol is raised and xanthomatous nodules appear in the skin especially on the backs of the elbows with xantho matous patches in the eyelids

**Course and Complications** The course is usually slowly progressive but there is a tendency to periodical febrile attacks with intensification of the jaundice Complications include intercurrent diseases such as erysipelas or pneumonia Coma occurs in the final stages and occasion ally there is ascites or gastric hæmorrhage

**Prognosis** The disease is usually fatal in about 5 or 6 years from its onset

**Treatment** This is similar to that required for portal cirrhosis Methyl testosterone tab mg 25 1 t d s for a few days will relieve the itching

### Obstructive Biliary Cirrhosis

(*Charcot s Cirrhosis*)

**Etiology** Charcot s cirrhosis results from obstruction and infection of the bile ducts as by a calculus adhesions around the common duct or carcinoma of the head of the pancreas

most satisfactory of the former adhesions being formed between the omentum and the anterior abdominal wall In the latter type of operation the portal vein is anastomosed to the inferior vena cava thus establishing a portacaval shunt

*Rupture of œsophageal or gastric veins* This constitutes a medical emergency demanding prompt and highly skilled treatment Bleeding may be controlled by tamponade a Sengstaken inflatable œsophageal tube being passed into the stomach The rubber tube contains three pathways one for inflating the balloon in the œsophagus the second for inflating the balloon in the stomach which compresses the cardia and the third for feeding the patient A blood transfusion should be given using fresh blood Feeding is by intragastric or intravenous drip with 10 to 20% dextrose solution Morphine must not be administered and if drugs are required to calm the patient paraldehyde may be given as for hepatic coma (see p 101)

### Post necrotic Cirrhosis

**Etiology** Post necrotic cirrhosis or scarring of the liver follows widespread necrosis of liver cells as may occur after infective hepatitis or in those cases of acute massive liver necrosis due to poisoning from phosphorus carbon tetrachloride or cincophen, which do not die during the acute phase The reason why it occurs in some cases of hepatitis and not in others is unknown

**Pathology** The liver is small with nodules and depressions on its surface The nodules are caused by proliferating liver cells and the depressions are due to areas of connective tissue with collapse of the liver reticular structure Broad bands of connective tissue are formed

**Clinical Findings** This disease is not uncommon in young people following one or more attacks of infective hepatitis The patient feels ill is jaundiced has abdominal pain and may suffer from hæmatemesis or ascites The serum globulin is raised, and the alkaline phosphatase in the serum is also usually above normal

**Course** In some cases there is rapid deterioration, the condition resembling acute yellow atrophy

**Differential Diagnosis** The condition resembles that met with in portal cirrhosis but there is usually a history of infective hepatitis The jaundice is deeper than in portal cirrhosis

**Treatment** This is similar to that for portal cirrhosis

### Biliary Cirrhosis
#### (*Unilobular Cirrhosis*)

There are two types which will be described separately cholangiolitic biliary cirrhosis and obstructive biliary cirrhosis

### Cholangiolitic Biliary Cirrhosis
#### (*Hanot s Cirrhosis*)

**Definition** A disease of young people characterised by enlargement of the liver persistent jaundice and periodical febrile attacks

**Etiology** The cause is not known Alcohol is probably not a causative factor *Predisposing causes* 1 Age 20 to 30 years but it may occur in children 2 Sex It is more common in women

**Pathology** Post mortem the liver is enlarged with a smooth surface and dark green colour It is firm on section and a fine perilobular fibrosis is present The larger bile ducts are normal the smaller ones show cholangitis or pericholangitis The spleen is normal or enlarged The portal lymph glands may be enlarged

**Clinical Findings** The disease is very rare The onset is gradual the patient complaining of loss of appetite attacks of nausea or diarrhœa jaundice and itching of the skin Pain may be felt in the region of the liver or of the spleen

*On Examination.* The patient is jaundiced but not usually very deeply The liver is found to be enlarged and may extend from the fourth rib to 4 inches below the costal margin It feels smooth and firm The spleen may be palpable There is no ascites The fingers are often clubbed and hypertrophic osteo-arthropathy may be seen in the wrists or other joints The blood There is a hæmolytic anæmia but the leucocytes are often increased up to 12 000 per c mm. or more The van den Bergh reaction is indirect The urine This contains urobilin and increased urobilinogen The fæces Stercobilin is diminished

**Differential Diagnosis** It may be confused with acholuric jaundice Banti s disease portal cirrhosis infective hepatitis and xanthomatous biliary cirrhosis The characteristic features are the persistent jaundice of a rather mild degree the absence of ascites and the enlarged liver The red cells show no increased fragility as they do in acholuric jaundice In Banti s disease there is usually a leucopenia and hæmorrhages occur before the onset of jaundice Portal cirrhosis is characterised by a milder degree of jaundice and the tendency to ascites In obstructive jaundice the fæces contain little or no bile In xanthomatous biliary cirrhosis the blood cholesterol is raised and xanthomatous nodules appear in the skin especially on the backs of the elbows with xanthomatous patches in the eyelids

**Course and Complications** The course is usually slowly progressive but there is a tendency to periodical febrile attacks with intensification of the jaundice Complications include intercurrent diseases such as erysipelas or pneumonia Coma occurs in the final stages and occasionally there is ascites or gastric hæmorrhage

**Prognosis** The disease is usually fatal in about 5 or 6 years from its onset

**Treatment.** This is similar to that required for portal cirrhosis Methyl testosterone tab mg 25 1 t d s for a few days will relieve the itching

### Obstructive Biliary Cirrhosis

(*Charcot s Cirrhosis*)

**Etiology** Charcot s cirrhosis results from obstruction and infection of the bile ducts as by a calculus adhesions around the common duct or carcinoma of the head of the pancreas

**Pathology** At autopsy the liver is usually contracted The surface is irregular and the colour is dark green On section it presents a honeycomb appearance due to dilatation of the bile ducts Fibrosis is present around the bile ducts, and this fibrosis may be unilobular or multilobular Calculi may be present in the extrahepatic bile ducts The spleen is not usually enlarged

**Clinical Findings** The onset is gradual with progressive jaundice

*On Examination* The liver is not generally palpable but an enlarged gall bladder may be felt The blood The van den Bergh reaction is direct The urine Bile pigment is present The fæces are pale owing to absence of bile

**Differential Diagnosis** The diagnosis involves consideration of the causes of obstructive jaundice, especially that due to gall stones The onset of cholæmic symptoms indicates that there is further failure of hepatic function

**Course and Complications** The course is progressive unless the obstruction is removed before irreparable damage has been done to the liver

**Prognosis** Early treatment may effect a cure The outlook is very grave with the onset of coma

**Treatment** The obstruction should be removed if possible by operation

### Other Varieties of Cirrhosis

These include pigmentary cirrhosis parasitic cirrhosis syphilitic cirrhosis and congestive cirrhosis

*Pigmentary cirrhosis* This is exemplified by hæmochromatosis (see p 676)

*Parasitic cirrhosis* This is due to infection of the liver with such parasites as the Bilharzia mansoni or the liver fluke (fasciola)

*Syphilitic cirrhosis* In congenital syphilis a pericellular cirrhosis may be noted In acquired syphilis single or multiple gummata are found in the liver Deep scarring in a healing gumma may produce the *hepar lobatum*

*Congestive cirrhosis* This is due to heart failure Fibrosis occurs chiefly around the central hepatic veins

## Jaundice

### (*Icterus*)

**Definition** A condition characterised by excess of bile pigment in the blood with a yellow discoloration of the skin and conjunctivæ

**Physiology and Pathology** Bile pigment is made from red cells engulfed by the phagocytic reticulo endothelial cells in the bone marrow spleen and liver The reticulo endothelial cells in the liver (Kupffer cells) are situated in the walls of the intrahepatic branches of the portal vein The bilirubin, which is the globin free iron free fraction of hæmoglobin is removed from the blood in the liver by the epithelial (polygonal) cells and excreted *viâ* the bile canaliculi to the bile duct In the intestines the bilirubin is reduced by the action of micro organisms into stercobilinogen, which is the same as hydrobilinogen or urobilinogen



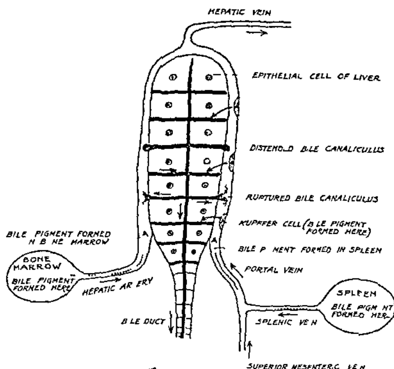



FIG 8 DIAGRAM (MODIFIED FROM RICH) SHOWING PART OF A LIVER LOBULE

If an excess of bile pigment is brought to the liver by the portal vein and hepatic artery and the liver cell function is simultaneously depressed excess of bilirubin leaves the liver by the hepatic vein and there is hepatic jaundice If the liver cells are necrosed or the bile ducts are obstructed bile pigments and salts regurgitate into the radicles of the hepatic vein and obstructive jaundice results

This is a colourless compound The greater part is probably oxidised in the large intestine to stercobilin which is the same as hydrobilin or urobilin This is a brown substance and colours the fæces A small amount of stercobilinogen is absorbed into the blood and carried by the portal vein to the liver The greater part of the absorbed stercobilinogen is re-excreted in the bile either as bilirubin or stercobilinogen but a trace may pass from the liver by the hepatic veins and be excreted from the general circulation in the urine as urobilinogen which is oxidised to urobilin when exposed to the air When the liver is damaged the stercobilinogen which is absorbed from the intestine by the portal vein to the liver is not recovered by the liver and re excreted in the bile but passes to the general circulation and is excreted in the urine in pathological amounts as urobilinogen and urobilin This implies that in addition to the liver damage a certain amount of bile pigment is reaching the intestines and so any obstruction, if present is incomplete

*The Classification of Jaundice* None of the classifications propounded can be accepted with enthusiasm and this is inevitable owing to the inadequacy of present knowledge The old division of jaundice into obstructive and non obstructive groups has the merit of being non committal as regards the nature of the second group McNee s classification into obstructive toxic and infective hepatic and hæmolytic cases subdivides the non obstructive group but is liable to criticism The nomenclature is mixed being based both on pathogenesis and on etiology Further such sharp divisions probably never occur in practice In extra hepatic obstruction of the bile ducts unless only of short duration there is probably always some intrahepatic damage and in hæmolytic jaundice there is also usually a hepatic element Arnold Rich has proposed a classification based on clinical laboratory findings and theoretical views as to pathogenesis He recognises two main types of jaundice 1 *Retention jaundice* In these cases there is excessive production of bile pigments by the reticulo endothelial cells and diminished excretion of bile by the liver cells The latter is due to subnormal function caused by various factors such as anoxæmia febrile disease or immaturity of liver cells The excess of bile pigment does not pass through the epithelial cells of the liver and enters the general circulation by the hepatic vein (see Fig 8) 2 *Regurgitation (obstructive) jaundice* The bilirubin in the blood is not excreted normally either owing to necrosis of the liver cells or to obstruction in the bile canaliculi or ducts The ducts then rupture (see Fig 8) and bile passes back into the blood channels of the liver *A combined form of jaundice* may also occur in which there is an excessive production of bile pigments and a regurgitation of bile which has been excreted into the canaliculi

### Obstructive Jaundice

The obstruction may be extrahepatic or intrahepatic

**Extrahepatic Obstruction** *Causes in the bile duct* These include a gall stone a round worm plugs of mucus a hydatid cyst or a projection of a carcinoma arising in the gall bladder *Causes in the wall of the duct* A new growth stricture or catarrhal inflammation *External*

*pressure on the duct* Carcinoma of the head of the pancreas, chronic pancreatitis, enlarged glands due to Hodgkin s disease malignant disease, and rarely tuberculosis or glandular fever or a gumma in the portal fissure carcinoma or ulcer of the stomach or duodenum, renal or suprarenal tumours an aneurysm of the cœliac axis, hepatic or mesenteric arteries

## Surgical Jaundice

This is due to post hepatic obstruction the common causes being a calculus in the common bile duct adhesions or stenosis affecting the bile duct system carcinoma of the pancreas of the ampulla of Vater *or of the bile duct or enlarged lymph glands in the portal fissure pressing* on the bile duct The jaundice is persistent and pruritus is often a prominent feature The signs of obstruction are present Absence of bile from the fæces, presence of bilirubin and absence of urobilinogen in the urine The direct van den Bergh reaction is positive The alkaline phosphatase reading is usually over 30 King Armstrong units and the flocculation liver function tests are negative The intense itching can usually be relieved by the administration by mouth of methyl testosterone mg 25 tab 1 t d s for 3 or 4 days If after about 4 to 6 weeks the diagnosis is in doubt it is usually wise to advise a laparotomy

**Intrahepatic Obstruction** This may be due to cholangitis cirrhosis or carcinoma of the liver or to hepatitis or necrosis of the polygonal cells of the liver An obstructive type of jaundice has been met with in patients taking chloropromazine

The varieties of obstructive jaundice such as that due to carcinoma of the liver or pancreas or to gall stones etc are considered under their respective headings

*Clinical Findings* *The urine is dark brown owing to the presence* of bilirubin but contains no urobilin The fæces are clay coloured and free from stercobilin They may contain a trace of stercobilinogen The blood contains an excess of bilirubin and gives an immediate direct van den Bergh reaction Often the obstruction is only partial The motions are then pale but contain some stercobilinogen the urine contains bilirubin and a trace of urobilin and the blood still gives an immediate direct van den Bergh reaction but the total amount of bilirubin in the blood is less than in complete obstruction

## Toxic and Infective Hepatic Jaundice

This may be caused by chemical substances such as arsphenamine bismuth phosphorus chloroform gold cincophen trinitrotoluene and tetrachlorethane It also results from infections as in typhoid fever Weil s disease relapsing fever pneumonia infective hepatitis yellow fever and septicæmia It is met with in pregnancy right sided heart failure and cirrhosis of the liver

Clinical Findings These vary in accordance with the degree of *intrahepatic* obstruction of the bile canaliculi If there is no obstruction the urine contains no bilirubin or only a trace and there is usually a

small amount of urobilin The fæces are normal in colour and the blood contains a slight excess of bilirubin the blood may give a delayed direct van den Bergh reaction If there is in addition to the liver damage, some intrahepatic obstruction the urine contains a varying amount of bilirubin with urobilin the fæces are pale and the blood contains an excess of bilirubin The van den Bergh reaction may be immediate direct or biphasic

### Hæmolytic Jaundice

**Etiology** This may occur in acholuric jaundice in splenic anæmia in pernicious anæmia and in paroxysmal hæmoglobinuria It may also result from blood transfusion when the blood is incompatible or in association with Diphyllobothrium latum infestation Other causes include poisoning from sulphonamide drugs malaria blackwater fever hæmorrhagic septicæmia polycythæmia rubra sickle cell anæmia and Lederer s anæmia

**Pathology** The spleen is often enlarged and there is anæmia In *these cases the renal threshold for bilirubin is often raised so that the* blood bilirubin is over 3 mg /100 ml without bilirubin appearing in the urine The jaundice is usually slight

**Clinical Findings** The jaundice is not usually severe The urine may contain a trace of bilirubin but there is frequently a marked excretion of urobilin The fæces are dark brown and contain an excess of stercobilin The van den Bergh reaction is a delayed direct one In long standing or severe cases where there is more damage to the liver cells bilirubin appears in greater quantity in the urine and an immediate direct van den Bergh reaction is given by the blood

### Constitutional Hyperbilirubinæmia

This is a rare condition in which the patient is permanently slightly jaundiced and which may be present from birth It has been called by other names such as familial non hæmolytic jaundice There is no evidence of a hæmolytic anæmia the liver function tests are normal and the indirect van den Bergh reaction is positive The condition is of no serious significance and it is thought that it may be due to a raised hepatic threshold for the passage of bilirubin from the blood into the bile capillaries

Another similar group of cases may be due to an inborn or acquired error of metabolism in which the liver cannot adequately excrete bilirubin and also bromsulphthalein and the dye used in cholecystography In addition to the impaired bromsulphthalein excretion and the failure to visualise the gall bladder in the cholecystogram the direct van den Bergh reaction is positive

### Jaundice in New born Infants

(*Icterus Neonatorum*)

The following varieties are described —

*Mild Types* Physiological appearing during the second or third day of life and lasting for 1 or 2 weeks The hyperbilirubinæmia is of the

indirect variety and probably due to an increased formation of bile pigment, dependent *less upon polycythæmia than* on a diminished excretory power of bile pigment by the liver cells

*Severe Types* These may be due to congenital stenosis or absence of the bile duct, gall stones or congenital syphilis of the liver These cases conform to the regurgitation type of jaundice Infections of the umbilicus or in the intestines may cause severe icterus neonatorum Two types of infective jaundice are described that of Winckel, in which there may be hæmaturia and that of Buhl, *which may show* hæmorrhage from the stomach intestines or umbilicus Erythroblastosis fœtalis is an important cause of severe jaundice in the new born and will now be considered

### Erythroblastosis Fœtalis

This is a disease which may affect the fœtus or new born infant There may be generalised œdema of the fœtus called *hydrops fœtalis* or a macerated still born fœtus with cirrhosis of the liver In other cases there is *icterus gravis neonatorum with erythroblastosis* or there may be severe anæmia with slight jaundice, a condition called *anæmia hæmolytica neonatorum* Owing to destruction of red cells in the blood red cells are formed in excess in other sites than the bone marrow spleen and liver such as the kidneys and suprarenals This is called extra medullary erythropoiesis or erythroblastosis In the blood we find an excess of nucleated red corpuscles Erythroblastosis fœtalis is usually associated with the Rh factor The Rh factor is an agglutinogen that is a substance in red cells which reacts with its corresponding agglutinin *present in the plasma of an incompatible blood* so that *if a transfusion* of such blood is given to a person whose red cells contain this agglutinogen agglutination of his red cells occurs with subsequent hæmolysis This agglutinogen is present in 85% of people and they are called Rh positive The factor is called Rh because it is present in the blood of the Rhesus monkey If the father is Rh positive and the mother is Rh negative the child may be Rh positive, but not every child does inherit the Rh factor from the father If it does the red cells of the fœtus contain the Rh agglutinogen This may provoke the formation in the *mother s plasma of an* anti Rh agglutinin a process known as iso immunisation It is necessary to postulate that some of the red cells of *the fœtus pass into the maternal circulation* If these agglutinins now pass across the placenta into the fœtal circulation agglutination and destruction of the red cells of the fœtus will occur The first child of such a marriage is not often affected possibly because iso immunisation of the mother takes place slowly and it is only during the second or third pregnancy that sufficient agglutinin is formed to cause hæmolysis If the mother is transfused with Rh positive blood of the correct group she is liable to have a transfusion reaction at the first transfusion as she already has anti Rh agglutinin in her blood Erythroblastosis fœtalis is not always due to the Rh factor In about 4% of cases it occurs when the parents are of different groups the agglutinin in the mother s blood which damages the fœtal cells being either anti A or anti B

Usually such an agglutinin is absorbed but in a few cases it remains free to attack the red cells In other cases pathological jaundice of the new born may be due to toxoplasmosis

An additional feature leading to hæmorrhages in some of these cases is hypoprothrombinæmia The jaundice is present at birth or appears within 24 hours and the liver and spleen are enlarged Some infants recover spontaneously and some who survive subsequently develop nervous lesions due to jaundice of the striato pallidal portion of the brain (kernicterus) The manifestations of this include choreo athetosis extrapyramidal spasticity opisthotonos and mental deficiency

Treatment This consists in the intravenous or intramedullary transfusion of 60 to 80 ml of Rh negative Group O blood The trans fusion should be repeated daily for 2 to 3 days If such blood is not available and the condition of the infant is critical the mother s red cells washed and suspended in saline may be used for the transfusion The mother s milk may contain the agglutinin in high concentration and for this reason some authorities advise that the infant shall not be breast fed Exchange transfusion of Rh negative blood may be carried out during the first 6 to 9 hours This gives the infant an adequate supply of Rh negative cells does not increase the blood volume and is likely to prevent kernicterus developing A polythene catheter is inserted into the umbilical vein and by means of a 20 ml syringe with a three way stopcock the infant s blood is withdrawn from the vein and the Rh negative blood is injected alternately The donor s blood is concentrated before injection by withdrawal of plasma to a red cell content of about 6 millions per ml and a total amount of about 400 ml are injected For hypoprothrombinæmia vitamin K should be admini stered 1 ml (containing 5 mg ) of inject menaphthone (B P ) being injected intramuscularly daily If the condition is still deteriorating immediate splenectomy affords a hope of cure

## Infective Hepatitis

Definition. Infective hepatitis is a condition due to an infective agent in which there are inflammatory changes in the liver and which is often accompanied by jaundice It is now usually believed that what was formerly described as catarrhal jaundice was in reality infective hepatitis Epidemics of jaundice have been recorded for over 200 years especially in times of war

Etiology The icterogenic agent is believed to be a virus not transmissible to animals and spread either by naso-oral droplets or by contamination of water by infected fæces It may possibly be spread by flies Alcohol appears to be a predisposing factor

Pathology The disease is probably a systemic infection with a predilection for the liver Aspiration biopsy of the liver shows necrosis and autolysis of the cells of the hepatic lobules with cellular infiltration around the periphery of the lobules There may be proliferation of the bile ducts in the portal tracts In some cases a permanent cirrhosis develops in others there is a mild residual fibrosis which may ultimately resolve

indirect variety and probably due to an increased formation of bile pigment, dependent less upon polycythæmia than on a diminished excretory power of bile pigment by the liver cells

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vomiting Avomine mg 25 may be given before meals For hæmorrhage vitamin K should be injected intramuscularly in doses of 5 mg daily and for serious hæmorrhage a blood transfusion should be given Methionine has not proved to be of value in the treatment of infective hepatitis

## Homologous Serum Jaundice

**Definition** Jaundice resulting from the introduction into man of human blood serum or plasma containing an icterogenic agent

**Etiology** It has been suggested that homologous serum jaundice is due to the same virus that is believed to cause infective hepatitis of this there is no proof The difference in the incubation periods is opposed to this view although this might be explained by the belief that the viruses are introduced at different sites in the two diseases The icterogenic agent does not behave in many respects like a virus

**Pathology** The microscopical findings in the liver in specimens obtained by liver puncture in cases of serum jaundice are similar to those seen in infective hepatitis

**Clinical Findings** The incubation period is about 100 days Homologous serum jaundice may arise in a variety of ways of which the following are the most important —By the injection of yellow fever vaccine prepared with human serum as the result of the prophylactic inoculation with measles convalescent serum or plasma or mumps convalescent plasma It may also result from the use of syringes contaminated with infected blood and employed either for intravenous intramuscular or subcutaneous injections or for removing blood by venepuncture when a small quantity of the material in the syringe is sucked back into the vein as the needle is removed This latter group includes post arsenical jaundice occurring in venereal disease clinics jaundice following the injection of penicillin or Pentothal and occasional outbreaks of jaundice in clinics for the treatment of diabetes mellitus or arthritis or in sanatoria Clinically the illness closely resembles that described above as characteristic of infective hepatitis but some authorities have pointed out differences in the clinical features especially the occurrence of erythema multiforme stiff joints splenomegaly and the usually apyrexial course in serum jaundice

**Treatment** *Prophylactic* Human serum is no longer used in the preparation of yellow fever vaccine and this type of homologous serum jaundice has now ceased Jaundice due to the use of contaminated syringes can be eliminated by adequate sterilisation All glass syringes should be sterilised by dry heat at 160 F for an hour if feasible In any case syringes should be well washed out after use and boiled between each patient even when the syringe is only used for withdrawing blood

*Curative* This is the same as for infective hepatitis

## Acute Massive Liver Necrosis

(*Acute Yellow Atrophy of the Liver*)

**Definition** A disease characterised by progressive jaundice fever severe nervous disturbances and shrinking of the liver

**Clinical Findings** The mode of onset course and severity of the illness are variable Outbreaks of jaundice were reported in Wensleydale by *Pickles in 1930* by *Newman in Sussex in 1942* by Cookson in Gloucestershire in 1944 and during the 1939–45 war epidemics of infective hepatitis occurred especially amongst the troops in Syria N Africa and Italy Four modes of onset have been recognised 1 Insidious with anorexia and gastro intestinal symptoms low grade fever and jaundice appearing after 3 or 4 days 2 Febrile the temperature suddenly rising to 101 to 103° F Jaundice occurs 2 to 4 days later 3 Ambulatory the patient continuing with his work until he is noticed to be jaundiced 4 Hepatic without any manifest jaundice Occasionally there is urticaria morbilliform rashes or arthralgia The temperature usually falls to normal in 3 to 4 days and the liver gradually enlarges and becomes tender the *spleen is seldom palpable After* a pre icteric stage of 4 to 5 days the temperature may rise again as the jaundice appears The stools may or may not be clay coloured and there may be constipation or diarrhœa Recovery is usually complete in 6 to 8 weeks *Laboratory Findings The white cell count is normal or there is* slight leucopenia The sedimentation rate of the red cells is usually normal in the icteric stage, and raised in the pre icteric stage and during convalescence The van den Bergh test gives usually a positive direct reaction, falling to normal in 40 days or less The urine occasionally contains urobilinogen in the pre icteric stage later bilirubin is present Liver function tests such as the bromsulphthalein and flocculation tests and the galactose tolerance and the Takata Ara tests usually give positive results The alkaline phosphatase reading is raised to about 25 King Armstrong units The incubation period is about 30 days

**Complications** These include hæmorrhage into the skin lungs or intestines nervous phenomena such as meningitis encephalitis polyneuritis myelitis and upper motor lesions of the pyramidal and striatal types Death may result from progressive jaundice and coma or from cirrhosis and œsophageal hæmorrhage

***Differential Diagnosis*** *In the pre icteric stage infective* hepatitis must be distinguished from influenza gastro enteritis sandfly fever malaria meningitis acute appendicitis enterica group infections or an alcoholic hang over When jaundice appears Weil s disease *can be excluded by the leucocytosis and positive agglutination findings*

**Treatment** *Prophylactic* Gamma globulin 10 ml injected intramuscularly during the incubation period, appears to be of definite value in preventing or attenuating an attack

*Curative* The patient should go to bed at the onset and remain there until the temperature is normal the liver has returned to its accustomed size and is no longer tender there are no symptoms such as lassitude and nausea and the serum bilirubin has been normal for a week It is usually advised that the diet should contain about *120 G* protein 200 to 300 G carbohydrate and 40 to 50 G fat or more fat if the patient desires it A light diet with plenty of milk from which the top layer of cream has been removed is usually suitable Fluids up to 5 pints a day should be taken during the acute stages *For nausea or*

vomiting Avomine mg 25 may be given before meals For hæmorrhage vitamin K should be injected intramuscularly in doses of 5 mg daily and for serious hæmorrhage a blood transfusion should be given Methionine has not proved to be of value in the treatment of infective hepatitis

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### Acute Massive Liver Necrosis

*(Acute Yellow Atrophy of the Liver)*

**Definition** A disease characterised by progressive jaundice fever, severe nervous disturbances and shrinking of the liver

**Etiology** The cause is not known The acute necrosis may occur in association with chemical poisons such as chloroform neoarsphenamine, alcohol phosphorus, trinitro toluene and tetrachlorethane A few cases have resulted from the therapeutic administration of Atophan A case has been recorded after a *prolonged Trilene anæsthetic death* occurring 11 days later It is also met with in pregnancy and occasionally in such diseases as typhoid fever and influenza It has been shown experimentally that necrosis of the liver can be produced by certain poisons of chemical origin (toxipathic hepatitis) or by lack of a nutritive factor contained in protein (trophopathic hepatitis) *Predisposing causes* 1 Age The majority of cases occur between the ages of 20 *and 30 but it may develop at any age 2 Sex* In adult life females predominate

**Pathology** At autopsy the liver is small greenish in colour and the capsule is wrinkled On section it is soft and yellow due to excess of bile and red areas caused by extravasated blood or hæmangiomata may be present The changes are necrotic rather than atrophic and begin in the periphery or centre of the lobules In cases showing a tendency to recovery nodular hyperplasia of liver cells may be found and the necrotic areas may be replaced by fibrous tissue The spleen may be enlarged and soft and meningeal hæmorrhages may be present

**Clinical Findings** The patient *may be a pregnant woman* who is taken ill with symptoms resembling those of infective hepatitis In about 5 or 6 days however her condition becomes very much worse the jaundice deepens vomiting becomes intractable and headache is severe

*On Examination* The patient is restless muscular twitchings may be seen and the tongue is brown and dry The breath has a fishy odour The area of liver dulness is diminished and it may be completely *obliterated The pulse is rapid and the temperature is usually about* 99 or 100° F The pupils are often dilated and the plantar response may be extensor The bowels are constipated and the motions clay coloured Hæmorrhages may occur from various sites such as the stomach intestines kidneys or under the skin The urine is dark containing bile pigments and usually albumin and casts Leucine and tyrosine crystals are also present The blood shows a low alkali reserve *owing to the acidosis It gives an immediate direct* van den Bergh reaction

**Differential Diagnosis** At the onset the case resembles one of infective hepatitis but in a few days it is obvious that the patient is gravely ill Acute hepatic necrosis may then be confused with spirochetal jaundice but the liver is not diminished in the latter disease In phosphorus poisoning also the liver is usually normal in *size or* somewhat enlarged

**Course and Complications** In severe cases the patient soon becomes delirious and comatose The temperature may rise rapidly to 106 F or higher just before death In milder cases a subacute phase follows

**Prognosis** Death usually occurs in about 2 weeks *in acute cases* in subacute cases it may be delayed for several weeks Recovery may occur in the milder types of the disease

**Treatment** The patient must be kept quiet in bed and the acidosis controlled by means of alkalis dextrose and insulin The dextrose can be given by mouth ½ to 1 lb daily in a quart of orangeade or by rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose every 4 to 6 hours or by intravenous drip of 1 to 2 pints of normal saline containing 5% dextrose with 5 to 10 units of insulin twice a day Sod bicarb gr 60 to 120 should be given by mouth every 24 hours Plenty of fluids in addition should be taken such as water and barley water and ½ to 1 pint of milk daily Amino acid intravenous injections in the form of protein hydrolysates are worthy of a trial given as a solution of 5% casein hydrolysate at the rate of 40 drops a minute each pint being alternated with 500 ml of 5% dextrose solution in distilled water or normal saline according to the chloride output in the urine The bowels should be kept open daily with salines such as mag sulph gr 60 to 120 mane or with enemata For insomnia and restlessness bromides gr 10 to 30 t d s should be given Morphine must not be administered owing to the risk of toxic effects

## Hepatic Coma

This is a rare complication of hepatic failure but it is also sometimes met with after portacaval anastomosis in which the liver is short circuited It is considered to be due to failure of the liver to metabolise nitrogenous substances absorbed from the intestines and in some but not in all cases there is a high blood ammonium level

The exciting cause is thought to be an excessive intake of protein or the administration of methionine choline or ammonium chloride

**Clinical Findings** Hepatic coma is characterised by the following features A far away look in the eyes untidiness mental confusion generalised tremors apraxia on dressing muscular rigidity exaggerated deep reflexes and later coma In addition the breath may have a sweet or slightly fæcal odour the so called *fœtor hepaticus* and on asking the patient to hold out the arms with the fingers separated a flapping tremor may be noted in which there are periodic movements of flexion and extension at the wrist and metacarpo phalangeal joints

Hepatic coma is of grave significance in infective hepatitis but in cirrhosis hepatitis it may assume a chronic form

**Treatment** In an acute attack no nitrogenous food must be given and the patient should be fed on dextrose and fruit juices If he cannot swallow a 20% solution of dextrose should be given by intragastric drip 3 to 5 pints in the 24 hours with the addition of 2 G of potassium chloride After a few days a high carbohydrate low protein diet can be given with a little fat The protein can then be increased to 20 G on alternate days Morphine and barbiturates must not be prescribed to control restlessness but paraldehyde m 60–120 can be given by mouth or m 360 of paraldehyde in 3 fl oz normal saline may be injected into the rectum Aureomycin or Neomycin should be given to reduce bacterial activity in the intestines the initial dose being 2 G followed by 0 5 G six hourly for 5 to 7 days

### Tumours of the Liver

Simple and malignant tumours may arise in the liver The simple tumours include an adenoma angioma and teratoma The adenoma may be sufficiently large to be felt during life An angioma usually gives rise to no symptoms or signs and is discovered at autopsy, but occasionally spontaneous rupture occurs with intraperitoneal hæmorrhage Teratomata are rare The malignant tumours are either primary or secondary carcinoma or sarcoma The majority of cases are secondary carcinoma

### Primary Carcinoma

**Pathology** The following varieties are described A large growth distending the liver substance smaller secondary deposits may occur in the liver A diffuse growth which may be scirrhous A multiple nodular growth Cirrhosis carcinomatosis, in which the carcinoma *probably develops in association with hepatic cirrhosis* Secondary deposits may be found in glands, the lungs brain bones etc

**Clinical Findings** The onset is insidious the patient complaining of progressive weakness, with perhaps pain in the region of the liver Frequently the tumour is not sufficiently large to be felt There is usually no jaundice and often no ascites but the patient rapidly goes down hill and death occurs in a few months from the onset of the symptoms

### Secondary Carcinoma

**Pathology** At autopsy the liver is enlarged, and numerous deposits of whitish growth can be seen on the surface and on section The projections may be umbilicated in the centre owing to necrosis Perihepatitis may be present in association with the nodules The peritoneum may also be involved with ascites Dissemination to the liver is by the blood stream but with a primary growth in the breast the spread occurs by lymphatic permeation The primary growth is most frequently found in the stomach, then in the colon and rectum the œsophagus pancreas, gall bladder uterus breast lungs etc

**Clinical Findings** The patient is most commonly a woman over the age of 40 There may be a definite history pointing to a carcinoma of the stomach, rectum or breast or the first symptoms arousing attention may be hepatic in origin *In such a case it may be difficult* to detect the site of the primary growth The patient may complain of pains in the region of the liver in the back or down the arms The skin may also itch very violently although there is no icterus

*On Examination* The abdomen is usually distended, whereas the body generally is wasted and the patient appears cachectic The liver may be felt much enlarged and the umbilicated nodules on its surface may be palpable In other cases where there is ascites it may not be possible to feel the liver until the fluid has been removed The spleen is usually not enlarged Jaundice is not present in every case but when it occurs it is usually obstructive in type and progressive In addition to the ascites in the later stages there is œdema of the feet

and dilated veins are seen in the lower part of the abdomen A mass of growth may be felt near the umbilicus and enlarged glands may be present elsewhere according to the general dissemination There is usually a microcytic anæmia The temperature is often irregularly raised

**Differential Diagnosis** This is usually quite clear when the primary growth can be detected In other cases such causes of hepatic enlargement as cirrhosis of the liver *gummatosis amyloid degeneration* an abscess or hydatid cyst a stone in the common bile duct etc , must be considered The Wassermann reaction should always be determined and if found positive a course of anti syphilitic treatment should be given

**Course and Complications** The patient becomes progressively more ill and finally dies in the majority of cases with cholæmic symptoms There may be hæmorrhages in the skin and cholangitis may occur as a complication

**Prognosis** Death generally takes place within a year from the diagnosis

**Treatment** This is only palliative In some cases there is no pain in others it is very severe Relief can be obtained by aspirin gr 10 t d s by mouth by Nepenthe m 10 to 20 t d s by mouth or by subcutaneous injections of morphin sulph gr ¼ or of pethidine hydrochlor mg 100 as required

### Primary Sarcoma

This may form a single large tumour or be found as multiple nodules or as a diffuse growth It is very rare

### Secondary Sarcoma

The sarcoma is secondary to sarcoma of the adrenals the mediastinum the skin a bone or the uveal tract in the eye With melanotic sarcoma melanin may be found in the urine and the primary growth is in the skin or the eye

### Hodgkin s Disease

This disease is more fully described on p 532 When the liver is involved it is slightly enlarged and ascites or jaundice may develop The liver is firm and whitish in colour the lymphogranulomatous tissue being spread throughout its substance

### Amyloid Liver

**Etiology** Amyloid liver occurs in association with chronic pul monary tuberculosis syphilis and long standing cases of suppuration of the bones It is comparatively rarely seen

**Pathology** The liver is enlarged firm and smooth It is tough to cut and pale on section The amyloid degeneration is seen in the walls of the capillaries especially in the intermediate zone of the lobules

### Tumours of the Liver

Simple and malignant tumours may arise in the liver The simple tumours include an adenoma angioma and teratoma The adenoma may be sufficiently large to be felt during life An angioma usually gives rise to no symptoms or signs and is discovered at autopsy but occasionally spontaneous rupture occurs with intraperitoneal hæmorrhage Teratomata are rare The malignant tumours are either primary or secondary carcinoma or sarcoma The majority of cases are secondary carcinoma

### Primary Carcinoma

**Pathology** The following varieties are described A large growth distending the liver substance, smaller secondary deposits may occur in the liver A diffuse growth which may be scirrhous A multiple nodular growth Cirrhosis carcinomatosis in which the carcinoma *probably develops in association with hepatic cirrhosis Secondary* deposits may be found in glands the lungs brain bones etc

**Clinical Findings** The onset is insidious the patient complaining of progressive weakness, with perhaps pain in the region of the liver Frequently the tumour is not sufficiently large to be felt There is usually no jaundice and often no ascites, but the patient rapidly goes down hill, and death occurs in a few months from the onset of the symptoms

### Secondary Carcinoma

**Pathology** At autopsy the liver is enlarged and numerous deposits of whitish growth can be seen on the surface and on section The projections may be umbilicated in the centre owing to necrosis Perihepatitis may be present in association with the nodules The peritoneum may also be involved with ascites Dissemination to the liver is by the blood stream, but with a primary growth in the breast the spread occurs by lymphatic permeation The primary growth is most frequently found in the stomach, then in the colon and rectum, the œsophagus, pancreas gall bladder, uterus breast lungs etc

**Clinical Findings** The patient is most commonly a woman over the age of 40 There may be a definite history pointing to a carcinoma of the stomach rectum or breast or the first symptoms arousing attention may be hepatic in origin *In such a case it may be difficult* to detect the site of the primary growth The patient may complain of pains in the region of the liver in the back or down the arms The skin may also itch very violently although there is no icterus

*On Examination* The abdomen is usually distended whereas the body generally is wasted and the patient appears cachectic The liver may be felt much enlarged and the umbilicated nodules on its surface may be palpable In other cases where there is ascites it may not be possible to feel the liver until the fluid has been removed The spleen is usually not enlarged Jaundice is not present in every case but when it occurs it is usually obstructive in type and progressive In addition to the ascites, in the later stages there is œdema of the feet

**Differential Diagnosis** The absence of splenomegaly excludes Gaucher s disease and the Neimann Pick disease Neuro blastoma of the suprarenal with secondary deposits in the liver is rapidly fatal The negative Wassermann reaction excludes syphilis It may be impossible to distinguish von Gierke s disease from hypertrophic steatosis of the liver without a microscopical examination of a portion of the liver obtained by liver puncture or by excision.

**Course and Complications** The course is often prolonged but the child may eventually recover with return of the liver to its normal size although some degree of acetonuria is liable to persist

**Prognosis** If death does not occur from intercurrent infections such as enteritis or pneumonia there is a good prospect of recovery

**Treatment** No special treatment is available

## The Leukæmic Liver

**Etiology** The liver is usually affected in lymphatic leukæmia but less often in the myeloid type

**Clinical Findings** In addition to the general and local effects of the leukæmia the liver is enlarged but painless There is usually no ascites but jaundice may be present

## Tuberculosis of the Liver

**Pathology** The tubercle bacilli may gain access to the liver before birth by the umbilical vein or after birth through the hepatic artery portal vein or rarely by lymphatics The lesions may be miliary with pericellular cirrhosis or localised caseous nodules may be found

**Clinical Findings** It is not usually possible to diagnose hepatic tuberculosis during life

## Syphilis of the Liver

**Pathology** The lesions may be congenital or acquired *Congenital syphilis* The liver shows pericellular cirrhosis and in the late congenital type there may be gummata with fibrosis *Acquired syphilis* There may be hepatitis during the secondary stage and later multiple gummata may be found The gumma undergoes central necrosis fibrous tissue forms around it which on contraction and healing leaves a depressed scar (hepar lobatum) A multilobular cirrhosis may also occur

**Clinical Findings** In congenital syphilis the associated lesions are found elsewhere The spleen and liver are both usually enlarged and firm There is generally no ascites and often no jaundice In late congenital syphilis the enlargement and irregularity of the liver may not be manifest until after the age of 10 years In acquired syphilis of the liver there is often pain due to perihepatitis and a firm nodular liver is present The Wassermann reaction is positive

**Treatment** In congenital syphilis the treatment is as described on p 594 and in acquired syphilis as described on p 596

**Clinical Findings** *On Examination* The patient presents the signs of the disease which has led to the amyloid degeneration such as long standing pulmonary tuberculosis The *liver is enlarged and feels* smooth and firm There is no pain over the liver Ascites is not common

**Differential Diagnosis** The firm character of the liver helps to differentiate it from the painless enlargement met with in fatty *degeneration Other evidences of amyloid degeneration* may be found such as albuminuria diarrhœa and enlargement of the spleen

**Prognosis** This is grave, and amyloid degeneration is often the precursor of death

**Treatment** This is directed to the causative condition

### Fatty Liver

**Etiology** Fatty changes in the liver may be due to generalised obesity chronic alcoholism nutritional deficiencies chemical substances such as chloroform and phosphorus severe anæmias acidosis in diabetes mellitus or cyclical vomiting and in some cases to tuberculosis

**Pathology** The degenerating liver cell contents are not converted into fat The fatty changes may be due to lack of thiamine or of the lipotrophic factor In most cases there is transfer of fat from the tissue depots to the liver The liver is enlarged and greasy on section

**Clinical Findings** These vary with the causative factor The liver is often difficult to feel owing to its softness or to the obesity of the patient It does not cause pain

**Treatment** This must depend upon the nature of the causative condition

### Von Gierke's Disease

(*Hepatomegalia Glycogenica Glycogen Disease*)

**Definition** A disease of children characterised by enlargement of the liver acetonuria and hypoglycæmia

**Etiology** The disease results from a metabolic error which is probably congenital and is possibly due to the lack of a glycogen splitting ferment

**Pathology** The liver is enlarged due to excess of glycogen The kidneys *also and in some cases the heart and voluntary muscles may* show an accumulation of glycogen

**Clinical Findings** The patient is usually a young child who is brought to the doctor on account of swelling of the abdomen or attacks of faintness

*On Examination* The liver is much enlarged smooth and moderately firm The spleen *is not enlarged Distended abdominal veins may* be seen but there is no ascites The heart is sometimes enlarged owing to glycogen retention Acetone may be detected in the breath and urine There is no glycosuria In older children there may be evidence of infantilism The blood sugar is low and does not rise over 30 mg per 100 ml *in half an hour after* the injection of 1 ml of adrenaline The blood glycogen and cholesterol are raised

swollen The contents are clear turbid or bile or blood stained fluid and gall stones may be present The orifice of the cystic duct may be blocked by the swelling of the mucous membrane

**Clinical Findings** The patient complains of severe pain in the region of the gall bladder This may be paroxysmal and colicky in nature or a more continuous ache The pain may radiate all over the abdomen and to the right scapular region There may be intense nausea and vomiting

*On Examination* The right upper rectus muscle is on guard and tenderness is elicited over the gall bladder If there is cystic obstruction it may be possible to feel the distended gall bladder The temperature may be normal or slightly raised Usually there is no jaundice

**Differential Diagnosis** It is impossible to eliminate with certainty the presence of gall stones but in biliary colic due to calculi the pain is of a more excruciating nature An appendix abscess may closely simulate acute cholecystitis but the swelling in the former is usually lower in the abdomen With suppurative cholecystitis the constitutional disturbance is generally greater the temperature is higher and there may be sweating and rigors In pyelonephritis the typical urinary changes are found

**Course and Complications** An acute attack may rapidly subside but recurrence is not uncommon Complications include suppurative cholecystitis which may pass on to gangrene and perforation of the gall bladder Sequelæ include the formation of external adhesions which may produce pyloric obstruction and the development of chronic cholecystitis and gall stones

**Prognosis** This is always uncertain in any particular case

**Treatment** The patient should be kept in bed and pain relieved by the application of hot flannels over the gall bladder It may be necessary to inject subcutaneously morphin sulph gr $\frac{1}{6}$ to $\frac{1}{4}$ or pethidine hydrochlor mg 100 if the pain is very severe The bowels should be opened and the gall bladder encouraged to empty itself by giving mag sulph gr 60 to 120 in hot water fl oz 2 every morning A course of aureomycin or penicillin should be given The dosage of aureomycin is detailed on p 154 Penicillin should be injected intramuscularly in doses of 600 000 units of a procaine preparation morning and evening for 6 to 7 days The diet must be fluid during the acute stage such as milk and soda and meat extracts Operation is required in cases of suppuration

## Subacute Cholecystitis

(*Lipoid Cholecystitis Cholesterosis 'Strawberry Gall bladder* )

**Pathology** The gall bladder is usually normal externally but the cystic gland is enlarged Yellowish white spots are dotted all over the mucous membrane These are due to deposition of lipoid (cholesterol ester) in the cells of the mucosa Mulberry cholesterin stones may be present Streptococci are usually found in the submucous tissue and in the cystic gland

**Clinical Findings** The symptoms closely resemble those of catarrhal

### Actinomycosis of the Liver

**Etiology** The cause is the Actinomyces bovis (Streptothrix actinomyces) (see p 606)

**Pathology** The liver is usually infected secondarily to actinomycosis of the intestine The lesion presents a honeycomb appearance and is bright yellow There is often associated perihepatitis The infection may spread directly to the pleura or lung, into the peritoneum, or work its way through the abdominal wall

**Clinical Findings** The patient complains of ill health and malaise with fever In addition, there may be pain in the region of the liver

*On Examination* The liver is palpable and tender and the surface may be irregular The blood shows a leucocytosis If the actinomycotic lesion ulcerates through the abdominal wall the streptothrix will usually be found in the pus

**Prognosis** This is very grave

**Treatment** Penicillin, 1 000 000 units should be injected intramuscularly every 6 hours for about 6 weeks

## THE GALL-BLADDER

### Acute Cholecystitis

**Definition** Acute inflammation of the gall bladder

**Etiology** The inflammation results from bacterial infection The organisms most often found are the Bacterium commune (Bact coli) the Bacterium typhosum (B typhosus) streptococci and staphylococci Less frequently the Diplococcus pneumoniæ (pneumococcus) the Pseudomonas pyocyanea (B pyocyaneus) or anaerobes such as the Clostridium welchii (B welchii) may be present *Predisposing causes*
1 Age Usually over 40 years 2 Sex Females predominate
3 Constitution and habits Obesity, a sedentary life and constipation
4 Previous illness Especially typhoid fever and gall stones

**Pathology** The organisms may reach the gall bladder by various routes The blood Non hæmolytic streptococci may gain access to *the gall bladder by the cystic artery* coming from a distant focus in the tonsils or teeth The intestines The organisms are conveyed by the portal vein The bile duct The organisms such as the Bact typhosum *may ascend to the gall bladder against the bile stream The lymphatics* The organisms may pass from the liver to the gall bladder

Streptococci have been found in the submucous tissue of the gall bladder (mural cholecystitis) and *in the cystic gland in a high percentage* of cases, when they have not been present in the mucous membrane or in the contents of the gall bladder These are presumably organisms which have been carried by the blood from a *distant focus The* following varieties of acute cholecystitis are described Catarrhal suppurative phlegmonous gangrenous and membranous

### Acute Catarrhal Cholecystitis

**Pathology** The wall of the gall bladder is inflamed and adhesions may extend externally from its serous coat *The mucous membrane is*

is potent in an alkaline medium Two mixtures are ordered Hexamine gr 100 aq ad fl oz 1 and Pot cit and sod cit āā gr 100 aq ad fl oz 1 The patient begins the course with m 60 of the hexamine mixture and 1 fl oz of the alkaline mixture after breakfast tea and after a glass of milk or water last thing at night The hexamine mixture is increased by m 60 daily until the patient is taking fl oz 1 (gr 100) t i d The urine should be tested 3 times a day, as unless an alkaline reaction is maintained there is a risk of producing vesical irritability and hematuria This dose is continued for 5 or 6 weeks when the symptoms should have disappeared On waking mag sulph gr 30 to 120 should be taken in 2 fl oz of hot water an hour before breakfast and the patient should then lie on the right side This should cause the gall bladder to contract The amount of mag sulph taken is regulated by its effect on the bowels as it is not desired that there should be diarrhœa It is doubtful whether the sulphonamides or penicillin reach the gall bladder in sufficient amounts to produce a bacteriostatic effect if the cystic duct is blocked There is no necessity to give a fat poor diet if there is no evidence of cholelithiasis

### Chronic Empyema of the Gall bladder

This may be a sequel of acute cholecystitis The patient complains of pain and tenderness in the region of the gall bladder which may be palpable There is usually no fever

### Torsion of the Gall bladder

This is a rare condition resembling clinically an acute abdominal emergency It may result in gangrene of the gall bladder Treatment consists in cholecystectomy

### Parasitic Infections of the Gall bladder

These are rare At times the Ascaris lumbricoides Lamblia intestinalis Echinococcus granulosus (T echinococcus) or the Distoma hepaticum may be present in the gall bladder

### Tumours of the Gall bladder

*Simple Tumours* These are rare and cannot usually be diagnosed They include papilloma adenoma and fibroma

*Malignant Tumours* These include primary and secondary carcinoma and sarcoma The majority of cases are primary carcinoma

### Primary Carcinoma of the Gall bladder

Etiology The cause is not known but the growth is often associated with gall stones or chronic cholecystitis Other predisposing causes include —1 Age Usually over 45 2 Sex Females predominate in the proportion of about 4 to 1

Pathology The growth may be papillomatous and fungating or diffuse and infiltrating It is most often situated at the fundus It may spread directly to the liver into the bile ducts or form a sinus through

cholecystitis but are of a milder degree than those described under the acute catarrhal infection

**Treatment** The gall bladder should be removed

### Chronic Cholecystitis

**Etiology** Chronic cholecystitis is due to infection with similar types of organisms as described for acute cholecystitis It may develop insidiously or follow an acute attack

**Pathology** The wall of the gall bladder is often thickened with external adhesions In some cases, if a calculus is occluding the cystic duct, the gall bladder is distended Calculi are often present and the cystic gland is enlarged

**Clinical Findings** The patient complains of chronic indigestion the chief features of which are flatulence and epigastric distention after meals and periodical attacks of nausea or vomiting Pain may be felt also in the region of the gall bladder or in the epigastrium and may radiate around the chest and pass to the right scapular region There may be slight icterus with fever from time to time

*On Examination* The right upper rectus is usually slightly rigid as compared with the left On palpation in the region of the gall bladder the patient may experience pain on taking a deep breath and the breathing may be suddenly checked (Murphy s sign) This may be best elicited if the patient is examined standing and bending slightly forward A cholecystogram may show that the gall bladder does not fill or contract normally or the gall bladder when thus visualised by the X rays may be found to be tender on palpation There may be achlorhydria

**Differential Diagnosis** Chronic cholecystitis may be confused with a gastric or duodenal ulcer with gall stones with chronic appendicitis or with angina pectoris The pain referred from arthritis of the spine is also misleading The opaque meal affords valuable indication as to the presence of gastric or duodenal ulcers Gall stones may be shown by direct X ray or by the cholecystogram but they are usually associated with chronic cholecystitis

Chronic appendicitis also may be present with chronic cholecystitis but in chronic appendicitis palpation over the appendix when visualised by X rays usually causes pain An X ray examination of the mid thoracic spine will exclude arthritis

**Course and Complications** Chronic cholecystitis unless adequately treated is usually a progressive lesion An attack of acute cholecystitis may occur at any time Complications include the formation of gall stones with biliary colic pancreatitis arthritis phlebitis and myocardial degeneration

**Prognosis** Chronic cholecystitis is a cause of persistent ill health but is not usually a fatal disease

**Treatment** Any focus of sepsis in the teeth and naso pharynx should be removed If gall stones are present or if there is evidence of chronic appendicitis the gall bladder and appendix should be removed In other cases an attempt should be made to disinfect the contents of the gall bladder by means of hexamine which in the presence of bile

gall bladder especially if stagnant may be unduly rich in pigment cholesterol or lime salts The gall bladder wall is frequently infected even when its contents are sterile Thus in a series of cases of gall stones the gall bladder was infected in 70% the fluid contents in 40% and the stones in 30% of the cases Aseptic calculi are thought to occur apart from infection especially when the blood cholesterol is high as in pregnancy and in some cases of obesity and arteriosclerosis A solitary calculus may be found or as many as 14 000 stones may be present in the gall bladder The stones may be situated in the cystic duct or less commonly in the common bile duct and the extra or intra hepatic ducts

*Varieties* 1 The pure cholesterol stone This is usually solitary, oval or circular pale and very light It may be formed apart from sepsis

2 The laminated cholesterol stone containing layers of cholesterol and calcium bilirubin

3 Mixed gall stones containing cholesterol (80% to 98%) and calcium bilirubin These are soft before they are dried

4 Pure calcium bilirubin (mulberry stones) Small hard irregular stones

5 Calcium carbonate These stones are rare

Clinical Findings In some cases stones in the gall bladder give rise to no symptoms and are only demonstrated post mortem In other cases the symptoms are those described above as being typical of chronic cholecystitis If the stone becomes impacted in the cystic duct the gall bladder enlarges and is painful but there is no jaundice When the calculus enters the common bile duct biliary colic usually ensues According to Courvoisier s law the gall bladder is usually not distended in cases of jaundice due to a calculus in the common bile duct, owing to the presence of old cholecystitis whereas in obstruction of the common bile duct due to a growth the gall bladder is dilated In some cases there is persistent jaundice without pain If the stone is lodged in the ampulla of Vater forming a ball valve obstruction there are usually periodical attacks of fever and jaundice known as *the intermittent hepatic fever of Charcot*

*Biliary Colic* This may occur when the stone enters one of the biliary passages The onset of the symptoms is often sudden occurring frequently during the night The patient complains of excruciating pain which radiates all over the abdomen to the right scapular region and tip of the right shoulder It is paroxysmal the patient rolls about or doubles up in agony There is sweating and often vomiting Attacks of biliary colic not due to a gall stone are described These may occur after cholecystectomy They are thought to be due to achalasia of the sphincter of Oddi but in some instances are due to pancreatitis

*On Examination* It is not usually possible to feel the gall bladder but the right upper rectus is somewhat rigid The temperature may be normal or slightly raised The pulse is feeble but is not increased in rate Jaundice may be noted a day or so after the attack and stones may be found in the fæces Whether or not gall stones can be seen by X rays

the abdominal wall or a fistula into the colon It may perforate intra peritoneally Thrombosis of the portal vein may be found and secon dary deposits in the liver, or in lymph glands in the portal fissure above the clavicles, or in the anterior mediastinum Microscopically the growth may be columnar or spheroidal celled and colloid changes may be present

*Clinical Findings* The patient is often a woman over the age of 45 who gives a history suggestive of chronic cholecystitis or gall stones More recently she has noticed increasing discomfort or pain in the region of the gall bladder, with loss of strength

*On Examination* In the early stages nothing may be found but later the gall bladder is palpable, tender, and the surface is often irregular The liver may also be enlarged If there is pressure on the bile ducts either from the growth or from the enlarged portal glands there will be obstructive jaundice There may also be ascites and swelling of the legs Enlarged glands may be felt above the clavicles In the later stages cachexia is more marked and hæmorrhages may be seen under the skin A cholecystogram may reveal a filling defect in the gall bladder

**Differential Diagnosis** In the early stages the symptoms usually suggest cholecystitis or cholelithiasis Later it is often difficult to differentiate carcinoma of the gall bladder from a growth in the liver pancreas or bile ducts An opaque meal should serve to exclude carcinoma of the stomach

**Course and Complications** The course is usually rapidly progressive Such complications as perforation, fistula formation or suppurative cholangitis may occur

**Prognosis** The disease is usually fatal within 8 months from the onset of symptoms

**Treatment** This is only symptomatic and palliative, as operative removal of the growth is usually impossible

### Secondary Carcinoma of the Gall bladder

The gall bladder may be affected secondarily by direct spread from a growth of the stomach or colon or by metastases from growths else where These deposits are usually subperitoneal

### Gall-stones

(*Cholelithiasis*)

**Definition** Calculi formed in the biliary passages or gall bladder

**Etiology** The main factors in their formation are Infection, biliary stasis and hypercholesterolæmia *Predisposing causes* 1 Age Usually over 40 2 Sex Females predominate 3 Habits A seden tary life and over eating 4 Associated conditions Pregnancy chronic diseases of the heart or lungs constipation 5 Climate Especially the temperate zones 6 Heredity There is a familial tendency

**Pathology** Frequently cholecystitis precedes the formation of calculi A central nucleus consisting of organisms (not infrequently anaerobes) mucus or fibrin may be the starting point The bile in the

## THE BILE DUCTS

### Congenital Obliteration

This is often associated with cirrhosis of the liver and jaundice is present either at birth or a few weeks later The liver and spleen are usually palpable bile pigment is present in the urine but not in the stools Hæmorrhages may occur in the skin mucous membranes or internal organs The condition is not syphilitic Death usually occurs within a few weeks

Congenital syphilitic stricture of the bile ducts is rare

### Suppurative Cholangitis

**Etiology** Suppurative cholangitis may be associated with gall stones cholecystitis or obstruction of the extra hepatic ducts by a tumour It may occur as a complication of such diseases as pneumonia or enteric fever or be secondary to an abscess in the liver

**Pathology** Suppuration may be found in the extra hepatic bile ducts and the gall bladder The liver is enlarged and numerous small abscesses may be present

**Clinical Findings** The chief features are fever with jaundice and rigors The patient is very gravely ill and the liver is usually enlarged and tender

**Differential Diagnosis** It is often impossible to diagnose suppurative cholangitis from suppurative pylephlebitis It is distinguished from intermittent hepatic fever (see p 111) by the intervals of comparative health in the latter disease

**Course and Complications** The disease is usually progressive Complications include pancreatitis and pyæmia

**Prognosis** This is always very grave and the disease is fatal when abscesses form in the liver

**Treatment** An operation may enable the extra hepatic ducts to be drained

### Chronic Catarrhal Cholangitis

This may be associated with cholecystitis and gall stones or occur as a sequela of hepatitis The extra hepatic ducts in these cases are usually affected In cirrhosis of the liver the intra hepatic ducts may be involved

### Calculi in the Bile Ducts

A description of the effects produced by calculi in the bile ducts is given on p 111

### Tumours of the Bile Ducts

*Simple Tumours* These include a papilloma adenoma and fibroma They are all rare

*Malignant Tumours* Pathology Primary carcinoma of the extra hepatic ducts appears in the following sites in this order of frequency At the union of the common hepatic and cystic ducts at the lower end of the common bile duct in the common hepatic duct in the cystic

depends upon their calcium content A cholecystogram may reveal their presence as a lighter shadow in the opaque gall bladder when they are not demonstrable in a direct radiogram

**Differential Diagnosis** Biliary colic must be differentiated from renal pancreatic or intestinal colic hiatus hernia a tabetic crisis associated with colic and jaundice, the pain of coronary thrombosis of acute pancreatitis or perforation of a gastric or duodenal ulcer The excruciating paroxysmal pain with its characteristic distribution usually serves to differentiate

**Course and Complications** The attack may last from a few minutes to a few hours Recurrences are liable to occur Complications include perforation of the gall bladder ulceration into the small intestine with subsequent intestinal obstruction formation of various fistulæ such as a gastric duodenal intestinal colic or bronchial fistula or a cutaneous sinus Suppurative cholangitis or liver abscess may also ensue Intestinal volvulus acute or chronic pancreatitis, or carcinoma of the gall bladder or bile ducts may follow

**Prognosis** Death during an attack is rare but succeeding attacks are liable to occur and gall stones may form in the biliary passages after cholecystectomy

**Treatment** *Prophylactic* Chronic cholecystitis should be treated as described above If there is a tendency to hypercholesterolæmia the diet should be fat poor and eggs should not be eaten

*Curative* During an attack of biliary colic the acute pain must be controlled either by the subcutaneous injection of morphine sulphate gr ¼ and atropine sulphate gr 1/100 by the subcutaneous injection of pethidine hydrochlor mg 100 by the intravenous injection of atropine sulphate gr 1/100 or by the inhalation of chloroform Tab glyceryl trinitrat gr 1/100 should be dissolved under the tongue at the same time as the morphine is injected Milder attacks may be mitigated by placing the patient in a hot bath and giving by mouth tnc belladon m 80 repeated in an hour if necessary Subsequently the gall bladder may be removed or the patient may receive a course of treatment as for chronic cholecystitis There is no known method of dissolving gall stones

## The Post cholecystectomy Syndrome

After cholecystectomy for gall stones symptoms persist or develop in about 25% of patients and in a higher percentage after removal of the gall bladder for other causes The pain nausea and intolerance to certain foods may be due to calculi in the ducts inflammation of the gall bladder stump cholangitis hepatitis pancreatitis adhesions biliary dyskinesia or to an error in diagnosis before operation the pain being due to some other cause such as a hiatus hernia arthritis of the spine etc If the patient is not jaundiced intravenous cholangiography using a preparation such as Biligraphin will often demonstrate the common bile duct the right and left hepatic ducts and some intra hepatic ducts and thus afford valuable aid in determining the condition of the biliary passages

abdominal pain which increases in severity He vomits and the bowels may be freely opened at first but soon are constipated

*On Examination* The patient is usually found lying on his back with his knees drawn up the breathing is shallow Abdominal movement is absent over the affected part of the abdomen The abdominal wall is rigid either locally or generally, and tender to light touch The abdomen becomes distended The liver dulness may be diminished if gas has escaped from the alimentary tract No intestinal movements can be detected with the stethoscope The facial expression is drawn and anxious (*facies Hippocratica*) The temperature may be a little raised or sub normal but the pulse is frequent and of small volume The tongue becomes furred and dry A leucocytosis occurs in the course of a few hours Pneumococcal peritonitis occurs more often in girls than in boys

**Differential Diagnosis** Acute peritonitis may be mistaken for intestinal colic obstruction or hæmorrhage a ruptured tubal pregnancy acute appendicitis without perforation acute hæmorrhagic pancreatitis mesenteric thrombosis a tabetic crisis or hysteria The important features in acute peritonitis are the absence of abdominal movement, and the rigidity These may however not be very evident in a case in which the peritonitis has been present for many hours The temperature may also be subnormal The pulse is a good guide as it becomes pro gressively more frequent as the condition deteriorates and the tongue becomes more dry

**Course and Complications** If untreated death occurs in the course of a day or so with generalised peritonitis but there may be a short period of temporary improvement which is deceptive Localised peritonitis may become completely shut off by adhesions and heal spontaneously

**Prognosis** This has been much improved by the use of sulphonamides and antibiotics

**Treatment** Streptomycin chloromycetin or aureomycin should be administered intravenously in doses of 500 mg twice daily until gastro intestinal intubation can be discontinued It should then be given by mouth in doses of 500 mg every 6 hours for about 5 days Fluid and salt loss must be made good by continuous intravenous administration of saline Ileus is relieved by intubation of the stomach or intestine and operation is required to repair a perforated viscus

### Acute Tuberculous Peritonitis

**Etiology** Acute tuberculous peritonitis is generally secondary to tuberculosis of the abdominal glands the intestines or genital organs

**Clinical Findings** The onset may closely simulate that of typhoid fever the patient being taken ill with headache malaise and abdominal discomfort The bowels are loose or constipated

*On Examination* The temperature is raised but often the pulse is not proportionally frequent The temperature assumes the continuous type with morning falls and evening rises of a degree or so Gradually the abdomen becomes distended first with flatus and later fluid may

duct The gall bladder is dilated with growths in the cystic duct and in the common bile duct, but there is no jaundice in the former case The growth may spread directly into the pancreas In some cases the *growth originates in the gall bladder and* spreads down the lumen of the bile duct

**Clinical Findings** The patient is usually an adult male The onset of the disease is often insidious with jaundice which becomes more intense progressive weakness and irregular fever There is usually no pain

*On Examination* The growth cannot be felt but the gall bladder may be palpable Bile is usually present in the urine, and the fæces are pale ' Silver stools resembling in colour aluminium paint have been described in cases of carcinoma of the ampulla of Vater chronic pancreatitis and sprue

**Differential Diagnosis** It is often difficult to distinguish a tumour of the bile duct from carcinoma of the head of the pancreas and in other cases, from a stone blocking the cystic duct

**Course and Complications** The course is steadily progressive and cholæmia occurs as a terminal phase

**Prognosis** The patient usually dies in a few months

**Treatment** An exploratory laparotomy is usually performed to establish the diagnosis and in some cases it is possible to relieve the jaundice by means of cholecystenterostomy

## THE PERITONEUM

### Acute Peritonitis

**Definition** Acute inflammation of the peritoneum

**Etiology** Acute peritonitis results from bacterial infection The causative organisms include streptococci the Bacterium commune (Bact coli) staphylococci the Mycobacterium tuberculosis (B tuberculosis), the Diplococcus pneumoniæ (pneumococcus) the Neisseria gonorrhœa (gonococcus) the Bacterium friedlanderi (pneumobacillus) the Pseudo monas pyocyanea (B pyocyaneus) the Bacterium typhosum (B typhosus) and anaerobic organisms

**Pathology** The organisms usually reach the peritoneum from the alimentary tract, either through a spot of lowered resistance in the wall or through an actual perforation They may also gain access from the gall bladder, a liver abscess the uterus or through the Fallopian tubes In some cases they may be blood borne or enter through a wound of the abdomen Terminal peritonitis may occur in chronic nephritis The peritonitis may be localised, as around the appendix and in the pelvis or diffuse The inflammation may be fibrinous or exudative and adhesions usually form after removal of the exudate The exudate may be serous purulent or hæmorrhagic and contain gas There is usually intestinal paralysis Certain varieties are described according to the primary infecting organism Streptococcal staphylococcal and Bact coli infections

**Clinical Findings** The patient is suddenly seized with agonising

its apex medially disposed close to the foramen of Winslow The infection originates in the appendix and rarely from a perforated gastric or duodenal ulcer from a liver abscess or from suppuration in the thorax

3 *Left Anterior Intraperitoneal* The abscess lies between the left lobe of the liver and the diaphragm and anterior abdominal wall The pus is derived from a perforated gastric or duodenal ulcer or from a splenic hepatic or pelvic abscess

4 *Left Posterior Intraperitoneal* The abscess is in the lesser sac of the peritoneum the foramen of Winslow being closed by adhesions The abscess is derived from a perforated posterior gastric ulcer a splenic hepatic or pancreatic suppuration or from spread of infection in general peritonitis

5 *Right Extraperitoneal* Suppuration occurs between the diaphragm and the bare area of the liver The infection is due to suppuration

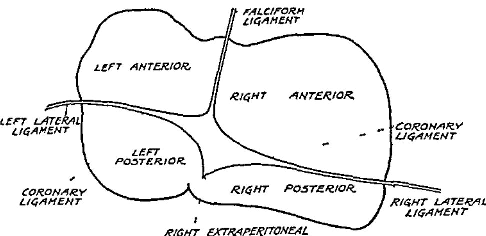



FIG 9 DIAGRAM SUPERO POSTERIOR VIEW OF LIGAMENTS OF LIVER SHOWING SITES OF SUBPHRENIC ABSCESS

in the liver or bile ducts or a spread from the abdominal wall by the lymphatics in the falciform ligament or from the right retroperitoneal tissue in a perforated posterior duodenal ulcer a retrocæcal appendix ulceration of the ascending colon or hepatic flexure or a perinephric or pancreatic abscess It may be secondary to suppuration above the diaphragm

6 *Left Extraperitoneal* Suppuration occurs in a potential space formed by stripping the peritoneum off the left side of the diaphragm The suppuration may be secondary to a left perinephric abscess a perforated posterior gastric ulcer ulceration of the colon diverticulitis acute osteomyelitis of the lumbar vertebræ perforation of the œsophagus or to suppuration above the diaphragm

Clinical Findings Subdiaphragmatic abscess is usually secondary to suppurative appendicitis or to perforation of the stomach or duodenum There may be a history of an operation from 4 to 7 months previously The temperature and pulse rate then rise and the patient

be detected Definite swellings due to matted omentum or glands may be felt

**Course and Complications** In a favourable case the temperature gradually falls to normal in the course of a few weeks complications such as a pleural effusion, intestinal obstruction tuberculous enteritis etc, may occur

**Treatment** The patient must be kept in bed during the febrile stage Streptomycin should be injected intramuscularly 1 G daily for 70 days, together with the oral administration of para aminosalicylic acid and/or isoniazid (see p 168) *Artificial or natural sunlight treatment* may be given starting with an exposure of 10 minutes daily to the feet and gradually exposing the whole of the legs for periods up to 1 or 1½ hours daily Paracentesis may be required if there is a considerable amount of fluid present

### Gonococcal Peritonitis

This is usually secondary to infection of the Fallopian tubes less often it occurs as a complication of gonococcal epididymitis The *peritonitis is generally localised to the pelvis* A course of a procaine penicillin should be given 600 000 units intramuscularly twice daily for 5 to 7 days

### Bile Peritonitis

This results from escape of bile which may follow cholecystectomy or be due to acute cholecystitis and necrotic lesions in the bile duct consequent on carcinoma or calculus In other cases the perforation may rapidly seal over and the peritonitis is then often due to rupture of a subserous duct on *the liver surface associated with cholangitis* The extravasation may at first be retroperitoneal and cause severe pain between the shoulders When it enters the peritoneal cavity it causes an outflow of plasma fall of blood pressure and shock The mortality rate is about 70% Adequate drainage affords the best hope of cure

### Subdiaphragmatic Abscess

*(Subphrenic Abscess)*

**Definition** A localised variety of acute peritonitis with suppuration *between the liver and diaphragm*

**Anatomy and Etiology** Six anatomical varieties are recognised according to the relation of the abscess to the hepatic ligaments four of these varieties are intraperitoneal and two are extraperitoneal (see Fig 9)

1 *Right Anterior Intraperitoneal* The pus collects between the right side of the diaphragm and the superior anterior and right lateral surfaces of the right lobe of the liver The infection is derived from an appendix abscess a perforated gastric or duodenal ulcer or from suppuration in the liver or bile ducts

2 *Right Posterior Intraperitoneal* (Cantlie s subhepatic pouch Rutherford Morrison s kidney pouch ) The pus collects in a pyramidal space, which lies transversely beneath the right lobe of the liver with

### Localised Plastic or Proliferative Peritonitis

**Etiology** Localised peritonitis may occur after abdominal operations or secondary to inflammation of the appendix gall bladder Fallopian tubes abdominal glands, diverticula etc In other cases it is a form of chronic tuberculous peritonitis Proliferative peritonitis may be localised around an organ such as the spleen or liver The latter may be covered with a thick white coat of fibrous tissue ('sugar iced liver see p 81)

### Diffuse Plastic or Proliferative Peritonitis

**Etiology** Diffuse peritonitis may occur as a form of chronic tuberculous peritonitis with carcinoma of the peritoneum or as an extension of a chronic plastic peritonitis of septic origin In Pick s disease there is a diffuse proliferative peritonitis often associated with similar proliferative changes in the mediastinum pericardium and pleura (see chronic indurative mediastino pericarditis p 221) and with chronic nephritis and arteriosclerosis In Concato s disease the peritoneum is much thickened the omentum is often rolled up and there is chronic perihepatitis or perisplenitis A serous effusion may be present and when the pericardium and pleuræ are involved the condition is known as polyserositis or polyorrhomenitis A barium meal may show a peculiar condition of thickening of the stomach and the cæcum and colon may also be involved

### Chronic Tuberculous Peritonitis

**Etiology** The Mycobacterium tuberculosis (B tuberculosis) usually gains access to the peritoneum from the intestine or the mesenteric and retroperitoneal glands It may however be a blood borne infection from a focus in any part of the body Cirrhosis of the liver is a predisposing cause in adults

**Pathology** Certain types are described which tend to overlap each other 1 *Tabes mesenterica* The mesenteric and retroperitoneal glands are affected 2 *The plastic or fibroid variety* Adhesions form the intestines are matted together the omentum may be rolled up in a mass and there is no effusion 3 *The ascitic form* The fluid may be free or loculated by adhesions 4 *The caseous variety* Softening occurs in the tuberculous foci there may be localised suppuration and the formation of fæcal fistulæ

**Clinical Findings** The patient is usually between the ages of 3 and 25 years The onset is insidious thus the child is gradually taken ill with lassitude loss of appetite, pallor abdominal discomfort or colic. The bowels are usually constipated

*On Examination* The child looks somewhat wasted but the abdomen is often distended The skin especially over the abdomen may be pigmented In the plastic variety the abdomen has a peculiar doughy feeling Small masses may be felt due to glands or matted omentum Loculated fluid gives rise to a dull swelling which may simulate an ovarian cyst When there is free fluid the signs are those of ascites

becomes more ill Rigors, sweating and pain in the region of the diaphragm or shoulder may develop

*On Examination* The patient looks ill If the abscess is anterior a bulging or swelling may be seen under the costal margin it is dull on percussion, and the chest moves little on the affected side If the stomach or duodenum have perforated, gas may also be present in the abscess and a resonant note may be obtained over a small area, which keeps uppermost as the patient is turned from his back to his side Abnormal signs are found at the base of the corresponding lung, for the diaphragm is raised and may be as high as the second or third rib Typically four zones can be detected at the back of the chest at the apex the normal lung below this is a zone of collapsed lung with some dulness and weak air entry still lower there is a tympanitic note due to gas in the abscess and at the base there is liver dulness When the abscess is a right posterior one a swelling may be found in the region of the right loin With a left posterior abscess there may be no swelling seen or one may be detected in the abdomen above or below the stomach With an extraperitoneal abscess on the bare area of the liver the liver is usually displaced downwards the right diaphragm is raised and there is dulness and deficient air entry over the lower lobe of the right lung The blood There is usually a leucocytosis of 15 000 to 20 000 per c mm An X ray examination may show the position of the diaphragm and of the liver Exploratory puncture through the 9th 10th or 11th intercostal space in the line of the vertebral border of the scapula may reveal the abscess the diaphragm being pierced by the needle before the pus is struck and so oscillating with respiration

**Differential Diagnosis** A subphrenic abscess is often difficult to diagnose it may be mistaken for septicæmia or pyæmia empyema pneumothorax, perinephric abscess liver abscess pylephlebitis or a pancreatic cyst The history X ray examination and results of explora tory puncture usually serve to establish the nature of the condition

**Course and Complications** If left untreated the abscess may cause a secondary pleural effusion or it may rupture into the pleura or lung, with resultant empyema or lung abscess Rupture may also occur into the pericardium œsophagus the general peritoneal cavity or through the skin *Septicæmia or pyæmia may occur as further complications*

**Prognosis** Subphrenic abscess is a serious condition with a mortality of about 70% if left undrained which is lowered by about a half if adequate treatment is given

**Treatment** The abscess should be drained surgically but the explora tory puncture should be performed in the theatre by a surgeon prepared to complete the operation The sensitivity of the causative organism should be determined and the appropriate antibiotic administered

### *Chronic Peritonitis*

**Definition** Chronic inflammation of the peritoneum

**Etiology** Chronic peritonitis may follow acute peritonitis or occur more insidiously The following varieties may be recognised —

glands in the portal fissure which may be due to tuberculosis carcinoma or Hodgkin s disease or the pressure of an aneurysm or thrombosis Nephritis pernicious anæmia or leukæmia *Seroperitoneum* may be due to Chronic peritonitis as in tuberculosis carcinoma or hydatid infection or to polyserositis or ovarian tumours *Hæmoperitoneum* may occur in peritonitis associated with tuberculosis or carcinoma *Chyloperitoneum* may be due to obstruction of or injury to the lacteals or to nephritis

Ascites is thus a sign of diverse pathological states

**Pathology** The fluid In hydroperitoneum the transudate is clear straw coloured with a specific gravity usually below 1 015 containing protein under 2% and a few cells In seroperitoneum the fluid may be darker the specific gravity is usually over 1 015 the protein content is higher (over 8%) and the cells are more numerous In hæmoperitoneum red blood corpuscles are present Chyloperitoneum may be chylous fluid whitish yellow and turbid with chyle a pseudochylous fluid may form in which the fat comes from degenerating cells Portal hypertension is the most important factor in the production of ascites in liver disease hypoproteinæmia is of great importance in ascites associated with generalised œdema

**Clinical Findings** The clinical picture varies according to whether the cause is cardiac hepatic renal etc The local symptoms and signs are as follows The patient complains of abdominal swelling heaviness or discomfort There may also be dyspnœa or palpitations with swelling of the legs

*On Examination* The abdomen appears fairly uniformly distended when the patient is lying with some prominence in the flanks provided the fluid is not loculated If there is a large quantity of fluid the umbilicus may be everted and striæ atrophicæ may be seen Enlarged veins may be present around the umbilicus (*caput Medusæ*) and with portal thrombosis dilated veins appear in the hypogastrium in which the direction of flow is reversed (*i e* the blood passing from below upwards) On palpation a fluid thrill may be obtained The thrill is transmitted to one hand placed on the abdomen in the flank when the other flank is flicked with the finger the ulnar margin of an assistant s hand should be used to compress the abdominal wall in the centre to prevent transmission of the thrill along the wall On percussion there is dulness in the flanks and hypogastrium and this shifts when the patient turns on his side the upper portion becoming resonant If the liver is enlarged it may be felt by suddenly pressing with the fingers over it ( dipping ) With small degrees of ascites the dulness may become apparent in the umbilical region by percussing the abdomen with the patient in the knee elbow position Loculated ascites may cause dulness in one zone of the abdomen which does not shift with change of position There may be œdema of the legs and scrotum and the urine may contain a trace of albumin

**Differential Diagnosis** Ascites must be distinguished from abdominal distention due to a full bladder an ovarian cyst a tumour or flatulent intestinal distention The bladder should be emptied with

The temperature is usually slightly raised and irregular but it may be normal The bowels are costive in the plastic variety, but with tabes mesenterica obstruction of the lacteals may result in diarrhœa with fatty stools

**Differential Diagnosis** Chronic tuberculous peritonitis may be mistaken for other causes of wasting cœliac disease chronic appendicitis regional ileitis, salpingitis an ovarian cyst abdominal carcinoma and cirrhosis of the liver or other causes of ascites Diagnosis is established in the ascitic variety by removing a specimen of the fluid and injecting it into a guinea pig, in which tuberculous lesions develop in 2 to 8 months An X ray examination which reveals calcified abdominal glands is in favour of the presence of unhealed tuberculous glands in the abdomen

**Course and Complications** The course is prolonged in the majority of cases Complications include intestinal obstruction or perforation fæcal fistula formation localised suppuration, and general dissemination by the blood stream

**Prognosis** There is usually a tendency to recovery, the most unfavourable developments are the general dissemination of tuber culosis intestinal obstruction the formation of fæcal fistulæ or of local abscesses

**Treatment** Streptomycin para aminosalicylic acid and/or isoniazid should be administered as for acute tuberculous peritonitis Operation is required for intestinal obstruction

## Cysts of the Peritoneum

The cysts may be mesenteric hydatid dermoid or teratomatous They cause vague symptoms such as abdominal discomfort and swelling and the treatment is surgical

## New Growths of the Peritoneum

These are simple and malignant Simple growths include a fibroma lipoma angioma lymphangioma and myoma Malignant growths may be primary such as an endothelioma or sarcoma Usually they are secondary to carcinoma of the ovary stomach intestine or breast or to a pleural endothelioma The peritoneum may be studded with minute nodules and an effusion serous hæmorrhagic or chylous may form

## Ascites

*(including Hydroperitoneum Seroperitoneum, Hæmoperitoneum and Chyloperitoneum)*

**Definition** Non purulent fluid in the peritoneal cavity

**Etiology** The fluid may be a transudate (hydroperitoneum) an exudate (seroperitoneum) blood stained (hæmoperitoneum) or fatty (chyloperitoneum)

*Hydroperitoneum* may result from Heart failure constrictive pericarditis or obstruction of the inferior vena cava Cirrhosis of the liver Obstruction of the portal vein as by pressure from enlarged

and mucus are passed by the intestine in a few hours the abdomen becomes distended and vomiting and perhaps hæmatemesis set in Gangrene or perforation of the intestine may occur The outlook is very grave, and the treatment is surgical

### Cysts and New Growths

Mesenteric cysts may be hæmorrhagic serous lymphatic or due to hydatid infection or to a dermoid They form rounded swellings in the abdomen and cause vomiting colic or intestinal obstruction

New growths are usually secondary malignant deposits

### Tuberculous Glands

(*Tabes Mesenterica*)

Tabes mesenterica is described on p 119 (chronic tuberculous peritonitis)

a catheter With an ovarian cyst the swelling is mainly below the navel the dulness does not extend to the flank or shift as it does with free fluid in the peritoneum and on vaginal examination displacement of the uterus may be felt With a tumour the mass if palpable, is generally firm and there is no shifting dulness With flatulent distention the abdomen *is hyperresonant but flatulent distention and ascites often* coexist

**Course and Complications** These must vary with the underlying cause of the ascites

**Prognosis** Ascites is almost always a serious condition in many instances being the harbinger of death The outlook is more favourable in cases due to congestive heart failure and a few cases of *benign ascites* have been described which have not recurred after paracentesis and for which no cause was discovered

**Treatment** The treatment for ascites due to cirrhosis has been *described on p 89*

## THE MESENTERY

The following affections of the mesentery will be briefly described Inflammation (mesenteritis) structural abnormalities, hæmorrhage thrombosis embolus, cysts, new growths, and tuberculous glands

### Inflammation
### (*Mesenteritis*)

This may form part of a general peritonitis or be localised to a part of the mesentery as the result of spread of infection from adjacent intestine

### Structural Abnormalities

*Excessive length of the mesentery may lead to volvulus* and the bowel may become incarcerated through an opening in the mesentery

### Hæmorrhage

This may occur rarely in the hæmorrhagic varieties of the acute specific fevers such as smallpox, or as the result of degenerative changes such as arteriosclerosis or aneurysm of the mesenteric arteries It produces symptoms resembling those of intestinal obstruction

### Thrombosis and Embolus

Venous thrombosis may result from cirrhosis of the liver and congestive heart failure or develop in association with pylephlebitis or intestinal or appendicular suppuration or after splenectomy It may also occur as a manifestation of thrombo phlebitis migrans An embolus may lodge in the mesenteric arteries in cases of infective endocarditis and mitral stenosis The clinical picture is that of an acute abdominal emergency The patient is suddenly taken ill with severe abdominal pain collapse sweating pallor low temperature and frequent pulse The abdominal wall is not usually rigid Some blood

and mucus are passed by the intestine in a few hours the abdomen becomes distended and vomiting and perhaps hæmatemesis set in Gangrene or perforation of the intestine may occur The outlook is very grave, and the treatment is surgical

### Cysts and New Growths

Mesenteric cysts may be hæmorrhagic serous lymphatic or due to hydatid infection or to a dermoid They form rounded swellings in the abdomen and cause vomiting colic or intestinal obstruction

New growths are usually secondary malignant deposits

### Tuberculous Glands

*(Tabes Mesenterica)*

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# CHAPTER II

# THE RESPIRATORY SYSTEM

**Introductory** Clinical investigations in diseases of the lungs include a routine examination of the chest by inspection, palpation percussion and auscultation *The lungs should be examined radio* logically with a screen and film and in some cases after the injection of Lipiodol or after establishing an artificial pneumothorax Tomography is of value in demonstrating deep seated cavities for by this process serial views of the lungs are obtained at varying depths from the surface Bronchoscopy and exploratory thoracotomy are required in some cases

*The vital capacity of the lungs* is estimated by a spirometer It is the amount of air which can be expired after a deep inspiration The average amount for an adult is 3 600 ml It is especially diminished in emphysema and is also found to vary with the degree of activity in pulmonary tuberculosis The sputum should be examined for colour consistency and odour it should be floated on water to see if bronchial casts are present and the volume in 24 hours recorded on the tempera ture chart Laboratory tests include a search for tubercle bacilli the predominating organisms and special tests for spirochætes ova of paragonimus westermani streptothriceæ and malignant cells It may be necessary to determine the Wassermann reaction of the blood the rate of sedimentation of the red cells and the cell count

## *THE UPPER RESPIRATORY TRACT*

### Hay Fever

(*Allergic coryza Pollinosis*)

**Definition** Paroxysmal and seasonal inflammation of the conjunctivæ and nasal mucous membrane

**Etiology** Hay fever is due to sensitiveness to certain pollens In England the pollen is usually that of the Timothy grass and more rarely tree pollens In the autumn the Michaelmas daisy is an occasional cause

**Pathology** The condition is one of allergy, the patient exhibiting an abnormal sensitivity to foreign proteins or to altered tissue proteins There is hyperæmia of the conjunctivæ of the nasal mucous membrane, and sometimes of the larynx and trachea

**Clinical Findings** The patient is usually a young adult who gives a history that every year in the early summer from May to July he suffers from attacks of sneezing watering of the eyes and often headache There may be much watery discharge from the nose and some malaise He may also suffer from asthma at different periods of the year The

attacks are more common in the country especially if near a hay field They also occur in towns

**Differential Diagnosis** The diagnosis is established by the seasonal incidence and the cutaneous reaction of the patient to pollen Hay fever should be differentiated from paroxysmal sneezing due to local irritation of the nose or to nervous influences (nervous coryza) In paroxysmal rhinorrhœa attacks of sneezing or of running from the nose occur at all times of the year This is probably a vasomotor phenomenon due to allergy the patient being sensitive to dust snuff orris powder feathers animal emanations bacteria etc

**Course and Complications** The course of the disease is limited by the life history of the plants producing the pollen Asthma or a perennial coryza may develop as complications

**Prognosis** This is good as subsequent attacks may be prevented or modified by appropriate treatment Hay fever tends to lessen in severity in successive years and to disappear in about 20 years

**Treatment** *Prophylactic* Early in the year the cutaneous reaction to Pollaccine should be determined A control prick is first made through a drop of normal saline with a hypodermic needle A drop of Pollaccine containing 20 000 units of pollen toxin per ml is placed on the forearm and a prick is made through it The drops are then wiped off with cotton wool A positive reaction is shown in about 15 minutes by the formation of an urticarial wheal surrounded by a red areola Desensitisation should be begun early in February Subcutaneous injections of Pollaccine are given every other day The initial dose varies from 40 to 100 units and a 15% increase is made at each injection In order to obtain adequate protection a final dose of 50 000 to 100 000 units will be required A local reaction may be prevented by taking calcium lactate gr 10 half an hour before the injection A marked local or general reaction can be checked by the subcutaneous injection of m 3 to 5 of liq adrenal hydrochlor Should a reaction occur the next injection should be the largest preceding dose that did not cause a reaction

*Curative* Considerable relief can often be obtained by the daily subcutaneous injection of 100 units pollen toxin and a subcutaneous injection of m 8 of liq adrenal hydrochlor Further treatment for the patient who is suffering from hay fever consists in wearing dark glasses The nasal mucous membrane may be protected by white vaseline or by Anesthone cream Privine or Fenox drops up the nostrils may give temporary relief Hydrocortisone snuff gives good immediate results Antihistamine drugs such as Histantin mg 50 tab Anthisan mg 50 tab or Dibistin mg 50 tab the latter being a combination of Antistin and Pyribenzamine may be helpful if taken 3 or 4 times a day

## Epistaxis

**Definition.** Bleeding from the nose

**Etiology** Epistaxis may be due to local or general causes *Local causes* Trauma picking the nose foreign bodies in the nose nasal diphtheria new growths such as a polypus or malignant tumour lupus

syphilis, telangiectases (which may be hereditary), a fractured base or a pituitary tumour *General causes* Epistaxis may be associated with high blood pressure mitral stenosis portal cirrhosis of the liver enteric fever, whooping cough, blood diseases such as leukæmia, anæmia hæmophilia and purpura, and a lowered atmospheric pressure such as occurs at high altitudes It may also occur at puberty without any discoverable cause

**Pathology** The bleeding point is often situated in Kiesselbach s area at the anterior and inferior part of the septum nasi

**Differential Diagnosis** Blood which passes backwards from the nose may be coughed up and raise the question of hæmoptysis A careful naso pharyngeal examination will usually reveal the bleeding point In other cases the blood may be swallowed and vomited or cause melæna

**Course and Complications** Usually the bleeding stops in a short time but it may be persistent and cause anæmia and debility

**Prognosis** This depends on the cause Epistaxis may be trivial, or it may be a symptom of a fatal disease

**Treatment** This varies with the cause In children the bleeding usually stops without treatment unless due to a foreign body, which should be removed In other cases a cold compress should be applied to the nose with the patient lying down or a little cotton wool inserted into the nostril and pressure applied over it The wool may be soaked in liq adrenal hydrochlor or in viper venom as described on p 549 In persistent cases the bleeding point should be sealed by the galvano cautery at cherry red heat Epistaxis associated with a high blood pressure is a useful safety valve and does not require to be checked unless very excessive

## THE LARYNX

### Acute Simple Laryngitis

**Definition** Acute catarrhal inflammation of the larynx

**Etiology** Acute simple laryngitis may be a symptom of a common cold, or result from inhalation of irritants It may also occur as an early manifestation of measles or be due to improper production or over use of the voice

**Pathology** There is hyperæmia of the vocal cords and epiglottis, with exudation of mucus

**Clinical Findings** The patient complains of alteration in his voice varying from harshness to hoarseness or aphonia The throat may feel raw and there may be malaise with an irritating cough

*On Examination* The temperature may be a little above normal and laryngoscopic examination will reveal the redness or swelling of the larynx.

**Differential Diagnosis** Acute simple laryngitis must be differentiated from functional aphonia and from such serious conditions as laryngeal diphtheria tuberculosis syphilis or carcinoma of the larynx which are described later

**Course and Complications** The laryngitis usually improves in

7 to 10 days but the course is more prolonged if the voice has been strained or if irritants have been inhaled

**Treatment** The voice should be rested If there is any fever the patient should be kept in bed the temperature of the room maintained at 65° F and the air moistened by a steam kettle A steam inhalation should be used for 5 minutes 3 times a day such as Ol pini m 10 mag carb lev gr 10 aquam ad m 120 M 120 in 1 pint of steaming water at 165 F Hot compresses applied to the neck are often comforting Cough can be checked by a sedative mixture such as Tnc opii camph m 20 tnc ipecac m 5 syr pruni serotin m 30 aq chlorof ad fl oz ½ Fl oz ½ t d s

### Chronic Simple Laryngitis

**Definition** Chronic catarrhal inflammation of the larynx

**Etiology** Chronic simple laryngitis may result from irritants such as tobacco and various forms of dust from over use or faulty use of the voice or from prolonged coughing associated with chronic bronchitis or pulmonary tuberculosis It may also be secondary to chronic infection in the mouth, nose or pharynx

**Clinical Findings** The onset is usually insidious the patient complaining of hoarseness or of weakness of the voice The throat may feel dry and there may be an irritating cough

*On Examination* The pharynx is usually red and swelling or redness of the vocal cords may be seen

**Treatment** The patient should rest the voice as much as possible and give up smoking and alcohol A steam inhalation should be used at night as described above and during the day a potassium chlorate lozenge may be sucked occasionally Subsequently lessons should be taken in voice production if the laryngitis has been caused by errors in this respect and a dusty occupation should be changed if possible In obstinate cases a change of air and rest may effect a cure

### Tuberculous Laryngitis

**Etiology** Tuberculous laryngitis is usually associated with pulmonary tuberculosis and is due to infection with the tubercle bacillus

**Pathology** The disease may be localised to the vocal cords inter arytenoid space ventricular bands or epiglottis There may be swelling infiltration or ulceration The posterior part of the vocal cords is most often affected

**Clinical Findings** Tuberculous laryngitis is very rarely seen now thanks to the antibiotic drugs The patient s first complaint may be of hoarseness or of loss of voice In other cases he may develop these symptoms as a complication of a recognised pulmonary tuberculosis If the epiglottis is involved the patient may notice difficulty in swallowing liquid foods any attempt to do so provoking cough In the later stages there may be severe pain on swallowing felt in the throat and radiating to the ear

*On Examination* Various lesions may be found in the larynx such as swelling or ulceration of the inter arytenoid space or of a vocal cord The vocal cord may be fixed by the granulation tissue

syphilis, telangiectases (which may be hereditary) a fractured base or a pituitary tumour *General causes* Epistaxis may be associated with high blood pressure, mitral stenosis portal cirrhosis of the liver, enteric fever, whooping cough, blood diseases such as leukæmia anæmia hæmophilia and purpura and a lowered atmospheric pressure such as occurs at high altitudes It may also occur at puberty without any discoverable cause

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## Malignant Tumours

**Pathology** Intrinsic growths occur in the larynx. The commonest variety is the epithelioma This may be a sessile tumour on a vocal cord or an infiltration of the tissues of the cord It may occur in other sites such as the ventricular band or the posterior commissure Extrinsic growths may be found in the epiglottis or around the cricoid cartilage Cordal carcinoma is very rare in women and when extrinsic laryngeal carcinoma appears in women it is nearly always situated centrally Secondary deposits in the cervical glands occur comparatively late in cases of intrinsic laryngeal carcinoma, but early in extrinsic growths Spheroidal celled carcinoma and sarcoma are both rarely seen

**Clinical Findings** The patient is usually a male over the age of 40 He first complains of hoarseness or of weakness of the voice often thought to be due to a cold but which persists despite treatment Later there is pain especially on swallowing and progressive deterioration of health due to cachexia from the growth

*On Examination* The growth is seen on laryngoscopic examination Later secondary deposits occur in the cervical glands and secondary infection of the growth may give rise to cervical cellulitis or abscess formation

**Differential Diagnosis** This can only be carried out by an expert laryngologist The difficulties are well known owing to the prominence given to the fatal illness of the late Kaiser Friedrich III

**Treatment.** This is surgical, or by X rays or radium

## Œdema of the Larynx

**Definition** Swelling of the larynx due to exudation of fluid

**Etiology** Œdema of the larynx may be inflammatory or non inflammatory *Inflammatory œdema* This may occur in association with acute catarrhal laryngitis septic pharyngitis cellulitis of the neck (Ludwig s angina) erysipelas of the face and neck ulceration of the larynx due to syphilis or tuberculosis or rarely as a complication of diphtheria scarlet fever typhoid fever etc

*Non inflammatory œdema* This may be a manifestation of angio neurotic œdema or be associated with cardiac or renal disease or it may result from hypersensitiveness to drugs such as iodides

**Pathology** The swelling affects chiefly the aryteno epiglottidean folds the vocal cords being only slightly involved owing to the tense attachment of their mucous membrane The epiglottis and subglottic region of the larynx may be very swollen

**Clinical Findings** The patient is often a child who is suddenly seized with dyspnœa and symptoms of asphyxiation There is a hoarse cry and inspiratory stridor The swollen epiglottis can usually be seen on oral examination

**Treatment** If the swelling is due to angio-neurotic œdema a subcutaneous injection of 0 5 to 1 ml of liq adrenal hydrochlor should be given immediately and an adrenaline spray may be used in an atomiser containing liq adrenal hydrochlor 1 part and normal saline 1½ parts If the œdema is due to other causes the immediate treatment

**Differential Diagnosis** Tuberculous laryngitis has to be differentiated from a simple laryngitis which may occur in pulmonary tuberculosis and from syphilis or a new growth The opinion of an expert laryngologist will be required In addition the sputum must be tested for tubercle bacilli the lungs X rayed the Wassermann reaction determined, and a portion of the diseased tissue may require micro scopical examination after removal by punch forceps

**Course and Complications** Tuberculous laryngitis may be cured by suitable treatment especially in the early stages Pulmonary tuberculosis is usually present

**Treatment** This is described on p 169

## Syphilis of the Larynx

**Etiology** Laryngeal syphilis is due to infection with the Treponema pallidum

**Pathology** *Congenital syphilis* A catarrhal syphilitic laryngitis *may occur in infancy or a gumma may develop about the age of puberty* *Acquired syphilis* Lesions may occur during the secondary stage such as patchy hyperæmia of the cords with formation of mucous spots In tertiary syphilis the lesions include infiltration serpiginous ulcera tion gumma formation or stenosis of the larynx The anterior part of the larynx is more commonly affected than the posterior

**Clinical Findings** There is usually no pain and no cough but the voice is hoarse or raucous Stridor is indicative of stenosis due to contraction of fibrous tissue

**Differential Diagnosis** This is as for tuberculous laryngitis The diagnosis is established by the laryngoscopic appearances the Wassermann reaction and the response to treatment

**Treatment** A full course of anti syphilitic treatment is required (see p 596) Caution must be exercised in the administration of iodides, as œdema of the larynx may ensue

## Tumours of the Larynx

These may be simple or malignant

### Simple Tumours

These include Papilloma fibroma singer s nodule (kerato dermia) angioma and degeneration cysts

**Pathology** Innocent tumours often grow from the vocal cords at the junction of the anterior and middle thirds of the cord A papilloma is frequently pedunculated

**Clinical Findings** The patient may have no symptoms but if the tumour is growing from a vocal cord there is usually hoarseness and there may be some stridor An angioma may give rise to hæmoptysis

**Differential Diagnosis** This is made by laryngoscopic examination and the microscopical appearances of the tumour after removal

**Treatment** This is surgical

Treatment *During the attack* The tongue should be pulled forward by passing the finger behind it and cold water sprinkled over the head and chest If this fails a little chloroform placed over the mouth on lint may cause the child to inhale *After the attack* The blood calcium should be estimated and if low calcium lactate given by mouth in doses of gr 1 t d s to a child of 1 year Rickets should be treated if present and enlarged tonsils and adenoids should be removed

### Infantile Laryngeal Stridor

This is due to a congenital deformity of the larynx in which the orifice is unduly small It disappears as the child grows

### Laryngeal Paralysis

**Introductory** The vocal cords are adducted on phonation and abducted with inspiration These movements are effected by intrinsic muscles The adductor group includes the lateral crico arytenoid muscles the inter arytenoid and the outer part of the thyro arytenoid muscles The abductors are the posterior crico arytenoid muscles The crico thyroid muscles also help to render the cords taut

Adduction is a specialised movement controlled by a cortical centre Abduction is mechanical and regulated by a centre in the medulla In functional paralysis adduction is affected, whereas in organic disease abduction is first lost *Semon s law* states that in a progressive organic lesion the intrinsic laryngeal muscles are affected in the following order Abductors tensors and adductors

The nervous path consists of a centre in the third frontal convolution of the brain and this is bilateral Thence upper motor neurones pass in the internal capsule to the lower motor neurone centre in the medulla (X and XI cranial nerve nuclei) The lower motor neurones run in the vagus the superior laryngeal branch of which supplies the crico thyroid muscle and the recurrent laryngeal branch innervates the other intrinsic muscles (the lateral and posterior crico arytenoids the inter arytenoid and the thyro arytenoids)

### Functional Aphonia

**Etiology** Functional aphonia occurs in association with hysteria shell shock debility or at times with pulmonary tuberculosis

**Clinical Findings** The patient speaks in a whisper but can cough normally There is no dyspnœa

*Laryngoscopic Examination* The cords appear normal at rest but on phonation adduction is incomplete Abduction is normal with inspiration

**Treatment** The general condition of the patient should be improved with a tonic such as syrup glycerophosph co m 60 t d s The voice may sometimes be immediately restored by local intralaryngeal faradic stimulation or by firm depression of the tongue with the middle finger of the right hand while the patient is told to cough and finish the cough on the sound Ah Suggestion is of great value in some cases The aphonia however, often recurs

consists in giving ice to suck, the application of ice cold compresses to the neck, and scarification of the œdematous epiglottis with a curved guarded bistoury after cocainisation In very severe cases tracheotomy may be necessary In cases due to iodide idiosyncrasy relief may usually be obtained by giving sod bicarb gr 60 t d s , and by omitting the iodides

### Croup

**Definition** A crowing form of respiration met with in infants and young children

**Etiology** The following varieties may be distinguished —

1 *Inflammatory* Laryngitis stridulosa Membranous laryngitis (diphtheritic and non diphtheritic) Simple acute laryngitis *Whooping cough*

2 *Reflex* Laryngismus stridulus Croup associated with enlarged tonsils and adenoids with dentition tetany and rickets

3 *Mechanical* Infantile laryngeal stridor Laryngeal polypi Foreign bodies in the larynx Pressure of an enlarged thymus upon the trachea

Certain of these varieties will be briefly described

### Laryngitis Stridulosa

**Clinical Findings** This is an affection of young children The symptoms of a cold are usually present with a cough and perhaps some hoarseness during the day The breathing is easy when the child goes to bed but he wakes up during the night with an attack of croup' characterised by cough inspiratory stridor and cyanosis This usually passes off in from a few minutes to half an hour when the child falls to sleep again

**Prognosis** The condition may be alarming but is not fatal

**Treatment** *If the attack does not rapidly subside the child should* be put in a hot bath A steam inhalation from hot but not boiling water should also be given (see p 127) or the nose and pharynx sprayed *with neb adrenal et ephedrin If these measures do not afford relief* an emetic dose of tinc ipecac should be given such as m 120 for a child of 6 years Subsequently if the tonsils and adenoids are enlarged they *should be removed*

### Laryngismus Stridulus

(*Spasmophilia Breath holding Attacks*)

**Etiology** This variety of croup may be a manifestation of tetany It is considered by some to be analogous to asthma and is often associated with rickets enlarged tonsils or dentition

**Pathology** There is laryngeal spasm but no inflammation

**Clinical Findings** The patient is usually an infant who is suddenly seized during the night with an attack of laryngeal spasm After some struggling the vocal cords relax and the air enters with a crowing sound Carpo pedal spasm may be present during the attack

**Prognosis** Death may occur during the attack

*Laryngoscopic Examination* Both cords are in the "cadaveric" position and they are immobile on phonation and on inspiration

## THE TRACHEA

### Tracheitis

**Etiology** Tracheitis may be acute or chronic *Acute tracheitis* This is usually bacterial in origin occurring either with a cold which extends to the large bronchi or in association with whooping-cough or influenza It may also be met with in measles diphtheria or enteric fever Inhalation of irritants such as poison gases or steam is a causative factor in some cases *Chronic tracheitis* This may follow acute tracheitis or be due to chronic irritation from smoking It may be secondary to chronic inflammation of the nose or larynx or to a local lesion in the trachea such as a tumour or gumma

**Pathology** The inflammatory changes vary in degree from the vascular engorgement exemplified by the 'pink trachea of influenza, to membrane formation with sloughing as may occur in diphtheria or gas poisoning

**Clinical Findings** In acute tracheitis the patient complains of a sense of soreness under the sternum but the symptoms of the associated laryngitis and bronchitis are usually more noticeable

**Treatment** In the acute stages the patient should be kept in a warm room and if there is fever he should be in bed Counter irritants should be applied over the upper part of the sternum and lower part of the neck such as lin camphoræ A sedative cough mixture should be given such as Tnc opii camph m 30 liq ammon acetat m 60 syrup pruni serotin m 30 aq chlorof ad fl oz ½ Fl oz ½ t d s A steam inhalation containing tnc benzoin co m 60 in a pint of steaming water at a temperature of 165 F should be used 2 or 3 times a day

### Tracheal Obstruction

**Etiology** The obstruction may be due to causes in the lumen of the trachea in the wall or outside the wall

1 *In the lumen* This may result from an inhaled foreign body, or from a pedunculated tumour such as a papilloma

2 *In the wall* Obstruction may be caused by cicatrisation of a wound or tracheotomy scar or by fibrosis following the inhalation of severe irritants Syphilis leprosy scleroma and secondary malignant deposits are rare causes of obstruction

3 *Outside the wall* Obstruction may result from pressure due to an enlarged thyroid ( scabbard trachea) enlarged cervical glands especially if affected by malignant growths or Hodgkin s disease an aneurysm an enlarged thymus or a mediastinal tumour

**Clinical Findings** These vary with the degree of obstruction and the suddenness of its onset The most noticeable symptoms are dyspnœa and tracheal stridor Pressure from an enlarged thymus in infants resulting in tracheal stridor is very liable to be mistaken for croup due to laryngeal obstruction The dyspnœa is often relieved by

## Organic Laryngeal Paralysis

**Etiology** The lesion may be in the brain, medulla vagus superior laryngeal or recurrent laryngeal nerve, or there may be a local laryngeal lesion, such as ankylosis of the arytenoid cartilage *A cerebral lesion* This must be bilateral to paralyse the vocal cords for stimulation of one centre causes adduction of both cords It is therefore very rare *A nuclear lesion* Involvement of the nuclei of the X and XI cranial nerves results in homolateral cord paralysis The chief causes are tabes dorsalis, a gumma, a hæmorrhage, a new growth disseminated sclerosis syringomyelia amyotrophic lateral sclerosis, poliomyelitis, and labio glosso pharyngeal paralysis These lesions are often bilateral *The vagus* This may be involved in a fracture or tumour of the base of the skull or by pachymeningitis or neuritis *The superior laryngeal nerve* This is rarely affected but it may be compressed by enlarged cervical glands or damaged by trauma or by diphtheritic neuritis Such a lesion causes loss of tension in the vocal cords *The recurrent laryngeal nerve* This may be compressed by an aneurysm glands in the neck, a thyroid tumour, carcinoma of the œsophagus chronic apical pleurisy especially on the right side a dilated auricle in mitral stenosis bronchial carcinoma a mediastinal tumour cervical or mediastinal abscess and a pericardial or pleural effusion It may also be affected by neuritis due to such causes as cold, diphtheria alcohol arsenic or lead It may be injured at an operation The following varieties of organic paralysis may be due to any of the causes given above

## Unilateral Abductor Paralysis

**Clinical Findings** There is no cough, the voice is normal or hoarse, and there may be some dyspnœa on exertion

*Laryngoscopic Examination* The cord lies near the mid line at rest it does not abduct on inspiration, but on phonation the cords meet

## Total Paralysis of One Cord

**Clinical Findings** The voice may be low and hoarse but there is no cough and usually no dyspnœa

*Laryngoscopic Examination* The cord is in the "cadaveric" position mid way between adduction and abduction *It does not* move on phonation or on inspiration On phonation the sound cord comes across the mid line to meet the paralysed one

## Bilateral Abductor Paralysis

**Clinical Findings** The voice is practically normal but there is inspiratory stridor

*Laryngoscopic Examination* Both cords at rest lie near the mid line They adduct on phonation but there is no abduction on inspiration Tracheotomy may be required to relieve the dyspnœa

## Bilateral Complete Paralysis

**Clinical Findings** The patient can only whisper, but stridor is not present

down to the chest There is malaise with perhaps headache and shivering as the temperature rises A sense of rawness may be felt under the sternum or in the second and third intercostal spaces near the sternum A dry cough causes pain in the chest which disappears as the secretion forms and is expectorated

*On Examination* The temperature may be raised to 100 or 101° F the respiration may be slightly increased to 18 or 20 and the pulse is also somewhat frequent 90 to 100 The chest Inspection Movement is good and equal Palpation Rhonchal fremitus may be present on both sides tactile fremitus is normal Percussion The note is normal Auscultation The breath sounds are harsh or they may be almost obscured by sonorous or sibilant rhonchi As the secretion loosens bubbling rales may be heard Vocal resonance is normal The sputum is scanty and tenacious at first later it increases and is more purulent

**Differential Diagnosis** There is usually no difficulty in the diagnosis either of the primary or secondary cases The sputum should be examined and the lungs X rayed to exclude the possibility of tuberculosis

**Course and Complications** The disease usually pursues a course lasting 2 to 3 weeks the temperature falling to normal in a week or so Chronic bronchitis may ensue as a sequela

**Prognosis** This is good unless the patient is very young or very old when the disease may prove fatal

**Treatment** The patient should be kept in bed in a warm and moist atmosphere the temperature being maintained between 60 and 65 F day and night and a steam kettle used In severe cases antibiotics should be given without awaiting the result of sputum culture Crystalline penicillin 1 million units should be injected intramuscularly every 12 hours Alternatively tetracycline (Achromycin) or chlortetracycline (aureomycin) can be given by mouth beginning with 1 G (4 capsules) and followed for 2 days by 0 5 G six hourly and then 0 25 G six hourly for about 5 days These drugs have a wide range of activity and are effective against the H influenzæ which is not the case with penicillin Vitamin B should also be given in the form of Beplex capsules 2 t d s During the dry stage a steam inhalation may be used such as Ol pini m 10 mag carb lev gr 10 aq ad m 120 M 120 in a pint of steaming water at 165 F The vapour to be inhaled from a Nelson s inhaler for five minutes night and morning For an irritating dry cough syrup codein phosphat (B P C ) may be given in doses of m 30 to 60 t i d Bronchial spasm may be relieved by the methods recommended for an attack of asthma Sleep may be induced by the use of some preparation such as Seconal gr 1½ capsule one or two or in some adult cases pulv ipecac et opii gr 10 The bowels should be opened daily with a saline such as mag sulph gr 60 to 120 mane During the febrile stage the diet should be liquid or semi solid As the secretion loosens and the cough becomes easier a stimulant expectorant should be given such as Ammon carb gr 3 tinc scillæ m 5 sp chlorof m 7 infus senegæ rec ad fl oz 1 Fl oz 1 ex aqua t d s If there are signs of dilatation of

leaning the infant forward The temperature is normal and the cyanosis and dyspnœa may improve markedly from time to time A foreign body which has passed through the larynx often gives rise to very slight symptoms when it is in the trachea and it usually rapidly passes into a bronchus In slowly developing obstruction the stridor is often heard first when the patient is asleep When the obstruction is severe, respiratory excursions of the larynx are obvious and the accessory respiratory muscles are in action

**Treatment** This varies with the cause of the obstruction Tracheotomy must not be performed in cases of thymic stridor, as it will only precipitate death The possibility of X ray treatment should be considered and the baby must not be allowed to lie down Foreign bodies in some cases can be located by X ray examination and removed with a bronchoscope *In organic stenosis in the cervical portion of the trachea* it may be possible to relieve the dyspnœa by a low tracheotomy Usually nothing can be done to relieve obstruction in the mediastinum due to external pressure unless there is a tumour which can be removed by operation

### Tracheal Diverticula

Tracheal widening with protrusion of its mucous membrane between the rings is a rare condition revealed at times by endoscopy or by Lipiodol examination

## THE BRONCHI

### Acute Bronchitis

*The following varieties are described Tracheobronchitis* suppurative and fibrinous bronchitis

### Acute Tracheobronchitis

**Definition** Acute inflammation of the larger bronchial tubes usually associated with tracheitis

**Etiology** *Primary cases* The exciting organisms are most often the Streptococcus pneumoniæ (pneumococcus) and the Hæmophilus influenzæ *Less often the organisms found are staphylococci strepto* cocci and Friedlander s bacillus Inhalation of dust and chemical irritants may also cause acute bronchitis *Predisposing causes* 1 Age Childhood and late adult life 2 Sex Males predominate 3 Climate Damp and wet 4 Season Autumn and winter 5 Heredity There is often a hereditary factor

*Secondary cases* Acute catarrhal bronchitis may occur as a complication in such diseases as measles whooping cough influenza enteric fever diphtheria nephritis, pulmonary tuberculosis malaria etc

**Pathology** The lower part of the trachea and the main bronchi are affected The mucous membrane is hyperæmic in the early stages, with little secretion later a thin mucous exudate appears which becomes muco purulent, and finally ceases

**Clinical Findings** *The patient is taken ill with a cold* which passes

ducts of the mucous glands dilate Later various changes occur such as purulent bronchiolitis minute abscess formation obliteration of bronchioles dilatation of other bronchioles and small areas of collapse and of emphysema

**Clinical Findings** The patient is often an adult male over the age of 40, who gives a history of recurrent bronchitis every winter He complains of cough shortness of breath and expectoration

*On Examination* There is often slight cyanosis of the face and the fingers may be clubbed The chest Inspection Movement is restricted especially if there is emphysema Palpation Rhonchal fremitus may be present and tactile fremitus diminished Percussion The note is hyperresonant and the area of cardiac and hepatic dulness is often encroached on by the emphysematous lung Auscultation The breath sounds are harsh or weak, and expiration is usually prolonged Scattered rhonchi or râles may be heard In some cases the adventitious sounds are only audible when the patient lies down, or after some exertion The vocal resonance is either normal or diminished Right sided cardiac dilatation may be present *Cough syncope* may occur causing temporary loss of consciousness This is due to cerebral anoxia resulting from the strain of coughing raising the intrathoracic pressure This reduces the return of blood to the heart and the cardiac output and blood pressure fall The sputum This may be scanty and in the form of sticky masses or it may be abundant resembling unboiled white of egg and water in the variety of bronchitis known as *bronchorrhœa serosa* In the majority of cases there is a small quantity of greenish black rather thick sputum and streaks of blood may be present from time to time In only about 15% of cases of chronic bronchitis is there X ray evidence of emphysema Bronchography shows some abnormality in 80% of cases the changes including abnormal variation of the bronchus calibre on respiration localised beaded bronchial dilatation bronchial diverticula and poor filling of the smaller bronchi

**Differential Diagnosis** This usually presents no difficulty Tuberculosis should be excluded by an X ray and sputum test and a bronchogram may be required in doubtful cases of bronchiectasis Early hypertensive left sided cardiac failure may be mistaken for chronic bronchitis

**Course and Complications** The course is progressive with seasonal intensification In the early stages the bronchitis is only present in the winter later it persists to a milder degree through the summer Complications include emphysema asthma bronchiectasis and dilatation of the heart

**Prognosis** This is usually unfavourable unless the course of the disease can be modified by climatic treatment

**Treatment** This is largely climatic If possible the patient should winter abroad in Egypt or South Africa Any septic focus in the naso pharynx should be removed In England an outdoor occupation or one involving the inhalation of smoke or dust is unsuitable The patient should give up smoking and if obese he

the right side of the heart cardiac and respiratory stimulants should be given such as digoxin mg 0 5 in 1 ml 70% alcohol diluted with 9 ml normal saline injected intravenously, slowly or Coramine 1 5 ml six hourly subcutaneously In a severe case 10 ml of Coramine may be injected intravenously If possible a period of convalescence should be arranged for, in a warmer climate

### Capillary Bronchitis

This variety of acute bronchiolitis is often indistinguishable from bronchopneumonia It may be associated with influenza, measles or scarlet fever Bronchiolitis fibrosa obliterans may develop later, nodules of fibrous tissue replacing destroyed bronchioles The X ray findings resemble those of acinonodular tuberculosis

### Acute Suppurative Bronchitis

An epidemic of acute suppurative bronchitis occurred in the winter of 1916–17 but the disease is seldom if ever met with now The patient is extremely cyanosed and dyspnœic the sputum being thick and copious The prognosis is very poor Oxygen should be administered with a double nasal tube or a B L B mask and crystalline penicillin injected intramuscularly one million units every 6 hours

### Acute Fibrinous Bronchitis

Some authorities regard this as a form of acute tracheobronchitis in which fibrinous casts form in the bronchial tree and are expectorated from time to time The casts can be detected by floating the sputum in water Attacks are liable to recur Steam inhalations should be given and crystalline penicillin 1 million units injected intramuscularly every 12 hours

### Chronic Bronchitis

**Definition** Chronic bronchial inflammation

**Etiology** Chronic bronchitis may be a recurrent hibernal sequela of an acute attack of bronchitis which was not efficiently treated or it may start insidiously The organisms most commonly found in the sputum are the pneumococcus H influenzæ staphylococcus aureus hæmolytic streptococcus and Friedlander s bacillus, any of which may be pathogenic Organisms of doubtful pathogenicity may also be present such as the N catarrhalis streptococcus viridans etc Tuberculous bronchitis is described on p 161 In some instances chronic bronchitis is secondary to cardiac or renal disease or to infection in the naso pharynx or cranial sinuses *Predisposing causes* 1 Age Usually over 40 2 Sex Males predominate 3 Climate Damp and fog 4 Season Late autumn winter and spring 5 Habits Over indulgence in alcohol and tobacco 6 Dusty occupations The professional classes are the least affected the unskilled workers the most

**Pathology** In the early stages there is hypertrophy of the goblet cells in the bronchioles and of the mucous glands in the bronchi The

bronchitis and with lung abscess Factors 1 and 2 are often combined but infection is the more important

3 *Congenital* This may be an error of development or due to atelectasis at birth In Kartagener s syndrome there is congenital dextrocardia and absent or incomplete development of the frontal sinuses Bronchiectasis associated with fibrocystic disease of the pancreas has been described on p 75

**Pathology** The bronchial dilatation may be 1 *Cylindrical tubular or rat tail* This dilatation is uniform or narrower at its termination 2 *Fusiform or glove finger* The dilatation is wider at its termination This occurs especially in a collapsed lung 3 *Saccular or globular* This resembles a bunch of grapes 4 *Moniliform or bead like* This suggests strung beads The left lower lobe is most often affected

**Clinical Findings** The patient is usually a child or a young adult although bronchiectasis may exist in elderly patients who are considered to be suffering from chronic bronchitis There may be a history of some antecedent causative condition such as pneumonia or bronchopneumonia The patient notices first that he has a cough and the sputum may contain small amounts of offensive material or it may be noticed that the breath is offensive after coughing or that the sputum or cough is influenced by change of posture This probably represents a pre bronchiectatic stage in which a bronchogram may reveal no abnormality The amount of sputum gradually increases and is more persistently offensive In other cases periodical unexplained rises of temperature with bronchitis may be the only symptom or there may be recurring hæmoptysis without any other symptoms (*forme hemoptoique seche*) In an established case the patient may say that life is unbearable owing to the amount of sputum and its horrible odour Such cases are rare now

*On Examination In an early case* The characteristic signs are the offensive nature of the sputum or of the breath but both of these may be absent The chest There may be slight dulness at one base with weaker breath sounds and a few persistent rales *In an established case* There is usually evidence of toxæmia as shown by wasting or stunted growth in a child cyanosis and clubbing of the fingers and toes The chest The signs are those of fibrosis and excavation Inspection Movement may be less on the affected side Palpation Vocal fremitus is diminished usually over the affected area The cardiac apex may be displaced to the side of the lesion Auscultation The air entry is usually weak over the bronchiectatic area and some leathery creaking or fibroid rales may be heard If there is a cavity which is comparatively empty and in communication with a bronchus the breathing may be bronchial or cavernous with bronchophony and whispering pectoriloquy Coarse rales may be heard If the cavity is full there is dulness with weak breath sounds and diminished voice conduction The sputum Expectoration is affected by change of posture Thus it may come up chiefly on rising in the morning or on lying down at night It is offensive The amount may vary from about 1 to 20 oz or more in the 24 hours On standing in a conical vessel it may settle into three layers frothy

should diet to reduce weight If the sputum is difficult to bring up the hot water medicine is helpful This contains Sod chlorid gr 3 sod bicarb gr 5 sp chlorof m 5 and aq anisi dest ad fl oz 1 Fl oz 1 in 1 fl oz of hot water on rising and repeated during the day if required If there is a looser cough an alkaline iodide mixture is helpful, such as Ammon carb gr 5 pot iod gr 3 pot bicarb gr 15 sp chlorof m 5 aquam ad fl oz 1 Fl oz 1 t d s Penicillin inhalations are of little value as the relief obtained is only temporary Tetracycline (Achromycin) or chlortetracycline (aureomycin) may be given by mouth in mg 250 capsules 2 capsules every 6 hours for a week and then 1 capsule every 6 hours for several months Vitamin B should also be given in the form of Beplex capsules 2 t d s If diarrhœa ensues the treatment must be discontinued For an acute exacerbation 1 million units of crystalline penicillin should be injected intramuscularly every 12 hours for a few days

If the disease is complicated by bronchial spasm antispasmodics should be given as for asthma, and if there is bronchiectasis daily postural drainage should be encouraged

### Chronic Suppurative Bronchitis

In this variety of chronic bronchitis the bronchial secretion is retained for a time in some of the bronchi and becomes offensive It may give rise to periodical unexplained rises of temperature and resemble the early stages of bronchiectasis

### Chronic Fibrinous Bronchitis

The patient, who is a sufferer from chronic bronchitis may from time to time have an attack of dyspnœa or a sense of constriction in the chest which is relieved by the expectoration of a fibrinous bronchial cast

### Bronchiectasis

**Definition** Dilatation of the bronchi

**Etiology** There are three main etiological factors 1 *Mechanical* Collapse of a bronchus either by partial obstruction from within or by pressure from without causes a fall of the intrapleural pressure with secondary dilatation of the adjacent bronchi resulting in bronchiectasis The causes include inhalation of a foreign body such as a bone a tooth a portion of a bath sponge a fragment of tonsil or adenoid tissue removed at an operation or plugs of mucus Pressure of an aneurysm tumour or calcified tuberculous glands may also cause collapse Pulmonary fibrosis associated with pleural adhesions may by traction lead to bronchial dilatation This may follow pneumonia or bronchopneumonia syphilis or tuberculosis of the lungs or be secondary to chronic pleurisy or penetrating chest wounds

2 *Infective* The bronchial wall may be weakened by chronic intrabronchial suppuration, and this together with a persistent cough may lead to bronchiectasis It may thus be associated with the inhalation of septic matter into the bronchi with chronic suppurative

above, turbid in the centre and a deposit below In the deposit the evil smelling Dittrich's plugs may be found Frequently the sputum is uniformly thick greenish pus The H influenzæ is often found in the sputum Hæmoptysis may occur from time to time The temperature chart may show rises corresponding with periods of retention of secretion, and falls when the expectoration is more profuse A direct X ray of the lungs may afford some suggestion of dilatation of bronchi which may be confirmed by tomography, but an X ray after intra tracheal injection of iodised oil will reveal the outline of the bronchi and thus establish the diagnosis

**Differential Diagnosis** The diagnosis usually lies between a condition of suppurative bronchitis, interlobar empyema a lung abscess, congenital cystic disease of the lungs bronchial carcinoma or pulmonary tuberculosis and various causes of recurrent pyrexia The bronchogram enables the diagnosis of bronchiectasis to be made

**Course and Complications** The course is usually progressive for a time and then a stationary stage is reached There is a great tendency for the other lung to become similarly affected Complications include septicæmia amyloid disease cerebral or spinal cord abscess empyema *pyopneumothorax suppurative pericarditis or gangrene of the lung*

**Prognosis** This has been much improved by antibiotic treatment but if the disease is firmly established and widespread the outlook is *very grave*

**Treatment** *Prophylactic* Breathing exercises to expand the base of the lung should be carried out during convalescence in every case *of pneumonia bronchopneumonia or empyema Great care should* be taken in operations on the nose and throat to prevent the inhalation of tissue from the operation site

*Curative* Cases should be *diagnosed early and medical treatment* instituted at once, thus avoiding the necessity of surgical operations Postural drainage as described below should first be tried Periodical courses of antibiotics should be given for 2 to 3 weeks, using *penicillin* tetracycline (Achromycin) or chlortetracycline (aureomycin) as in the treatment of chronic bronchitis Postural drainage without the administration of penicillin carried out regularly for *half an hour* 2 or 3 times a day may keep the bronchi dry The patient must be prepared to continue postural drainage indefinitely It can be carried out simply by lying face *downwards over a stool about 2 feet* high with the ribs clear of the seat of the stool the head hanging down and the hands on the floor A basin is put on the floor to receive the sputum as the patient *breathes deeply and coughs In more severe* cases tilting may be required for longer periods or by night as well as by day The correct position can only be determined after studying the bronchograms, the *object being to ensure that the pus in* the affected bronchi drains downwards into the trachea More than one position may be required to drain all the affected bronchi The foot of the bed can then be raised *on blocks or a special* Nelson bed used or more simply a metal bed elevator can be employed The foot of the bed

can thus be adjusted at different heights A mixture of creosote and potassium iodide may be given if the sputum is offensive such as Pot iod gr 5 creosot m 2 tinc quillai m 2½ ext glycyrrhiz liq m 20 syr tolu m 30 aq anisi dest ad fl oz ½ Fl oz ½ ex aq t d s p c In cases of bronchiectasis due to the inhalation of a solid foreign body bronchoscopy with removal of the causative object should be performed if possible Repeated bronchoscopic drainage does not afford much hope of a cure

Radical cure of bronchiectasis can usually only be effected by resection of the affected portion of lung This may imply segmental or lobar resection or total pneumonectomy In some cases removal of portions of both lungs may be necessary

### Bronchial Diverticula

Bronchial pouches resembling those described as tracheal diverticula (see p 134) are rarely seen

### Asthma

### (*Spasmodic Asthma*)

**Definition** Paroxysmal attacks of dyspnœa chiefly expiratory in nature associated with bronchial spasm

**Etiology** The allergic diathesis is an important factor There may also be an undue irritability of the broncho motor portion of the vagus nucleus or of the bronchial muscles themselves Emotional factors an over anxious parent domestic unhappiness worry and anxiety may all play their part in inducing an attack

*Spasm of the bronchi* may result from 1 Stimulation of the nucleus from psychical impulses such as emotions fatigue or the sight of artificial flowers 2 Reflex stimulation especially from the nose and at times from the eye stomach and intestines 3 Bronchial stimulation from the inhalation of cold air or fog 4 Injected substances such as acetyl choline or histamine carried by the blood to the bronchi 5 Inhibition of the sympathetic nerve supply as by endocrine influences 6 The effect of the adrenal corticoids is now assuming greater importance Ovarian asthma occurs in the premenstrual period and is due to lack of progesterone

*Œdema of the bronchi* may result from 1 Inhaled allergic substances such as pollens room dust book dust orris root powder animal emanations flower emanations drugs etc Printers asthma has been described due to inhalation of gum acacia in the spray fluid used in colour printing 2 Blood borne allergic substances such as digestive products of foods especially of eggs mill fish cheese etc drugs taken by mouth bacterial products liberated from foci of infection injected serums and skin testing materials

*Relaxation of the bronchi* may result from 1 Stimulation of the sympathetic nerve supply 2 Inhalation of substances such as stramonium and adrenaline 3 Blood borne stimuli such as adrenaline which has been injected or ephedrine taken by mouth (see Fig 10)

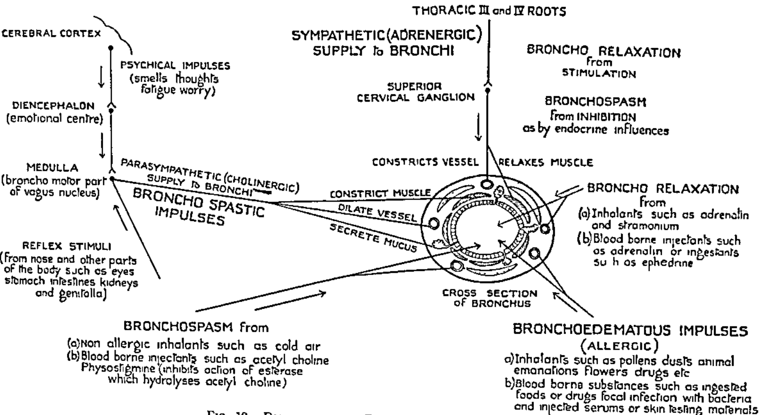



Fig 10 Diagram of the Paths of Impulses in Asthma

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patient is a subject of chronic bronchitis and suffers from periodical attacks of asthma Other causes of dyspnœa such as an enlarged thymus or thyroid or a loculated spontaneous pneumothorax compressing the upper half of the sound lung when the other one is extensively damaged by fibrosis may closely simulate asthma

**Course and Complications** The course in adults is usually progressive the attacks becoming more frequent unless some effective means are found for checking them In some cases an attack may last for 2 or 3 weeks a condition designated as the *status asthmaticus* In children asthma may cease spontaneously for no apparent reason Complications include emphysema bronchitis and dilatation of the heart Subcutaneous emphysema is a rare complication probably due to rupture of distended marginal vesicles Pulmonary tuberculosis may be associated with asthma Asthmatics are especially liable to anaphylaxis and serum should not be administered for the treatment of other diseases without preliminary desensitisation on account of the danger of the patient passing into status asthmaticus and dying within 24 hours

**Prognosis** Death during an attack is not so uncommon as was at one time thought Asthma tends to shorten life owing to the complications and is a very crippling disability Death may occur from right heart failure spontaneous pneumothorax or from suffocation due to blockage of the small bronchi by mucus

**Treatment** *During the attack* At the first symptom m 2 to 4 of liq adrenal hydrochlor should be injected subcutaneously or a Neo Epinine (isoprenalin sulph) tablet mg 20 dissolved under the tongue Adrenaline can also be administered as an endobronchial application A very fine nebulisation is required which is effected by a special spray or inhalation apparatus using a 1% aqueous solution One of the proprietary antispasmodics such as Riddobron or Rybarvin often gives relief when inhaled With some patients a tablet of ephedrine hydrochloride gr ¾ taken by mouth will ward off an attack If no relief is obtained other remedies should be tried such as placing a hot water bottle to the feet and drinking a cup of strong coffee or taking elix caffein iodid m 60 in water An inhalation of amyl nitrite m 5 from a capsule sometimes gives relief When the patient is acutely ill and has not obtained relief from adrenaline good results sometimes ensue from the intravenous injection of Cardophylin (aminophylline) 0 24 G in 10 *ml* sterile water Alternatively *Cardophylin may be injected* intramuscularly 0 48 G in 2 ml sterile water together with 2 ml 2% procaine Cardophylin may also be given by mouth 0 1 G tablet, 1 or 2 t i d In very severe cases it may be necessary to give an inhalation of chloroform For the milder attacks anything which distracts the mind may afford relief Thus one of my patients said to me The best cure for my asthma is a game of Bridge especially when I win For the *status asthmaticus* a syringe should be filled with 2 ml of liq adrenal hydrochlor and 0 1 ml injected subcutaneously every 5 minutes keeping the needle in position the attack may be terminated in some cases in half an hour by this method Som patients suffering

*Predisposing Causes* 1 Heredity Asthma often runs in families the allergic diathesis being inherited 2 Sex Males predominate 3 Age Attacks usually start in childhood or early adult life

**Pathology** During the attack there may be constriction of the bronchial muscles with expiration and in addition, hyperæmia or œdema of the mucous membrane with an increased output of mucus at the end of the attack The lungs become overdistended as the air enters comparatively easily but is expelled with difficulty This leads to emphysema Patients do sometimes die in an attack of bronchial asthma and on post mortem examination the bronchi may be filled with tenacious exudate Polyarteritis nodosa is found in some cases There may be secondary emphysema and dilatation of the heart

**Clinical Findings** The patient may give a history of eczema in childhood, or of a severe attack of bronchitis or bronchopneumonia which has been followed by asthma In a typical case the attacks occur by night, the patient going to bed apparently well and waking about 2 a m with shortness of breath He sits up in bed or may try to get near an open window There is great distress Asthma may recur at week ends This may be due to uncongenial home surroundings or personal relationships

*On Examination During an attack* The patient usually sits forward with the head resting on the hands and the elbows on the knees The face is pale and the expression anxious or alarmed. There may be cyanosis of the lips and ears The breathing is laboured short and jerky inspiration being followed by very prolonged and feeble expiration The accessory respiratory muscles come into play The chest Inspection The movements described above will be noted Palpation Rhonchal fremitus may be felt Percussion The note is hyperresonant and the cardiac and liver dulness may be diminished Auscultation Expiration is prolonged rather high pitched and wheezy and scattered rhonchi may be heard Vocal resonance is usually diminished The pulse is rapid and often weak during the attack The blood may show an eosinophilia up to 50%

The attack usually stops gradually with cough and some expectoration In the sputum Curschmann s spirals may be found consisting of small coiled threads of mucus with some leucocytes and eosinophil cells Octahedral Charcot Leyden crystals formed of spermin phosphate may also be present in the sputum The attack may last for 2 hours or longer and the patient remain wheezy for 1 or 2 days After the attack he often falls asleep exhausted *Between the attacks* There may be no abnormal signs found in the lungs but in a long standing case there is usually emphysema and the chest is deformed the shoulders being high and square and the upper dorsal spine kyphotic

**Differential Diagnosis** Other types of paroxysmal dyspnœa must be excluded Cardiac asthma There is usually a lesion of the heart with or without congestive failure The breathing tends to be less laboured but more rapid Renal asthma There is usually a history of chronic nephritis the urine contains albumin and relief is obtained by giving alkalis to overcome the acidosis Bronchial asthma The

individuals vary so much but the majority of asthmatic patients are free from attacks at an altitude of over 4 000 feet An asthmatic patient who is definitely emphysematous will probably be unable to tolerate such an altitude

## Bronchial Obstruction

**Etiology** The obstruction may result from causes within the bronchi changes in their walls or pressure from without 1 *Within the bronchus* The obstruction may be due to thick mucus to an inhaled foreign body to substances entering through a tracheotomy wound or to a gland ulcerating through the bronchial wall 2 *In the wall of the bronchus* Tuberculosis or syphilis may cause fibrosis or a scar may result from a foreign body which has been removed or from inhaled irritants Adenoma and bronchial carcinoma are important causes Muscular spasm is a factor causing obstruction in asthma 3 *Outside the bronchial wall* Pressure on the bronchi may be exerted in the mediastinum by enlarged glands a tumour an aneurysm an abscess carcinoma of the œsophagus or by a pericardial effusion Intra pulmonary tumours or an interlobar effusion may also cause bronchial obstruction

**Pathology** Bronchial obstruction leads to varying degrees of collapse of the pulmonary territory If the obstruction is due to a septic foreign body, then bronchitis bronchopneumonia bronchiectasis pulmonary abscess or gangrene may ensue An inhaled foreign body is rather more likely to pass to the right lung than to the left as the junction of the right bronchus with the trachea is slightly wider and at a less acute angle than is that of the left bronchus

**Clinical Findings** If the patient has inhaled a foreign body which lodges in a bronchus he usually complains of cough and perhaps a little discomfort in the chest A variable latent period may now occur before septic symptoms appear Often the first symptom noted is that the breath is offensive after coughing and then a little offensive sputum is brought up The patient becomes more acutely ill with cough expectoration and fever

*On Examination* A small area of dulness and bronchial breathing with a few fine rales may be found The physical signs in any individual case vary with the degree of obstruction and the amount of sepsis Thus a foreign body such as an acorn may obstruct the orifice of the main bronchus going to one lung The whole lung will then collapse with con sequent diminution of expansion on that side of the chest weak or absent air entry increased voice conduction and displacement of the heart to the affected side In other cases signs of pulmonary abscess or of bronchi ectasis develop The chest should be X rayed and in this way a solid foreign body may be revealed A bronchogram will indicate the site of bronchial obstruction If this is intrapulmonary and not due to a foreign body bronchoscopy will probably be necessary to decide whether the bronchial obstruction is due to an intrabronchial cause such as a primary carcinoma of the bronchus or to an intrapulmonary but extra bronchial cause such as a tumour of the lung

from the status asthmaticus are not relieved even by repeated injections of adrenaline In these cases oxygen should be given by the B L B or plastic mask at the rate of 6 to 9 litres a minute An alternative form of treatment consists in the intravenous administration of aminophylline (Cardophylin) as described above Morphine or pethidine may result in respiratory failure and should not be used In intractable asthma rapid relief may be obtained by the intramuscular injection of ACTH mg 25 6 hourly, or cortisone tab, mg 25 4 tabs 2 or 3 times a day for 4 or 5 days The dose should then be gradually reduced If sleep is prevented by fear of an attack phenobarbitone gr ½ to 1 should be given at night Amesec, which contains aminophylline, ephedrine *and amytal 1 capsule at night may also be helpful* *Between the attacks* In all cases of asthma it is advisable to examine the patient and not merely his skin reactions A search for a septic focus should first be carried out with investigations of the throat cranial sinuses and antra teeth fæces and urine The lungs should be examined radiologically to determine the degree of emphysema and to exclude tuberculosis The vital capacity should be estimated The sputum should be examined for tubercle bacilli and for the predominating organisms When the sensitivity of the organism has been determined the appropriate antibiotic should be given for 1 to 2 weeks An investigation should be carried out as to the relation of the asthma to locality foods, seasons animals flowers plants and injections The benefit obtained in some cases by a holiday in Switzerland or a week end by the sea may be due rather to the change of society than to the change of locality The cutaneous protein reactions should be determined Many cases of asthma are due to room dust or feathers and some to face powders and cosmetics The bedroom should be sparsely furnished with no carpet no covering to the chairs and the lightest of curtains Kapok pillows bolster and quilt and a rubber mattress should be used The room should be kept scrupulously free from dust with a vacuum cleaner and a damp cloth

In *premenstrual asthma* the patient should be given an intramuscular injection of 1 ml of Synergon (progesterone mg 10 and œstrone mg 1) every other day for 5 doses beginning 7 days before the period is due and repeated each cycle for a few months In bronchial asthma a course of potassium iodide and stramonium should be given such as Pot iod gr 3 tnc stramon m 10 ext glycyrrhiz liq m 20 sp chlorof m 5 aq ad fl oz 1 Fl oz 1 t d s This should be taken for a *month and then continued once or twice daily if it affords relief* In cases of allergy other than bacterial it is best if possible to avoid the substances to which the patient is sensitive Face and talcum powders free from orris root are obtainable An enquiry should be made as to whether or not the patient is sensitive to aspirin If he can take aspirin without cardiac disturbance *1 or 2 tablets at night will sometimes* prevent an attack where other measures have failed Expiratory breathing exercises are of value in all cases of asthma and a course as described in the Asthma Research Council booklet should be carried out No rules can be laid down as regards locality for residence as

### Primary Carcinoma of the Bronchus

Etiology The cause is not definitely known but the incidence of carcinoma of the bronchus has greatly increased during the past 20 years It is thought that inhalation of irritants is an exciting cause particularly that resulting from cigarette smoking Statistics indicate that there is a grave danger of contracting the disease if an individual smokes over 20 cigarettes a day for a period of years The risk from pipe or cigar smoking is considerably less It has also been shown that the sex incidence of bronchial carcinoma in non smokers is equal As bronchial carcinoma occurs in non smokers smoking cannot be the sole cause The disease is more common in urban than in rural areas and a smoky atmosphere may contain a carcinogenic agent There is no definite evidence that tar dust petrol or diesel oil fumes are exciting causes Other causes which have been postulated include arsenic in nickel workers asbestos and the fumes connected with coal gas manufacture The radio active dust from certain mines is also carcinogenic *Predisposing causes* 1 Age The maximum incidence is between 40 and 55 years 2 Sex Males predominate

Pathology It is now generally agreed that primary carcinoma of the lung actually arises in a bronchus from an undifferentiated cell in the basal layer of the epithelium The tumour may be squamous celled an adeno carcinoma or an undifferentiated round cell spindle cell or oat cell' growth In about 75% of cases the tumour arises in a main stem bronchus and is then usually squamous celled the remainder originate in the small peripheral bronchi the squamous celled variety preponderating to a lesser degree Central necrosis and involvement of the pleura or thoracic wall tend to occur in the peripheral variety Tumours situated centrally may rapidly spread to the mediastinal tissues giving rise to a hilar or mediastinal type In other cases diffuse infiltration of lung tissue may occur

Clinical Findings The patient is usually a middle aged adult who has suffered from a smoker s cough for some years This may have become worse recently In the early stages he appears in good health Wheezing may be an early symptom In the peripheral type especially a few streaks of blood or a small clot may be coughed up or pain in the chest may be complained of When the disease becomes more advanced certain clinical types can be recognised (*a*) *Dependent on the primary growth* 1 *Pulmonary* The chief symptoms are cough expectoration pain in the chest and dyspnœa There is loss of weight anorexia sweating and febrile attacks In some cases in which infection has occurred the onset is acute resembling influenza bronchopneumonia or lung abscess 2 *Mediastinal* The clinical picture resembles that of mediastinal pressure or of right sided heart failure There may be dyspnœa a brassy cough an asthmatoid wheeze cyanosis venous engorgement and œdema of the legs (*b*) *Dependent on the secondary deposits* The primary lung tumour remains small and symptomless and may not be revealed by X ray examination 1 *Nervous* Secondary deposits in the brain may suggest a primary cerebral tumour hæmorrhage or abscess encephalitis or meningitis Pressure of secondary

**Differential Diagnosis** There is usually little difficulty in diagnosis in cases of an inhaled foreign body A history of an operation on the nose or throat, which is shortly followed by cough with fetid breath and offensive sputum is typical of bronchial obstruction due to an inhaled septic foreign body Bronchial obstruction due to other causes is usually diagnosed by means of the investigations detailed above

**Course and Complications** These vary with the cause, the complications have been mentioned above

**Prognosis** A foreign body usually causes little trouble if it is a hard object which can be readily removed The outlook is serious with soft and septic foreign bodies The prognosis is grave with cases due to a new growth

**Treatment** A hard foreign body should be removed with a bronchoscope The treatment of the other varieties should be directed to the causative factor

### Tumours of the Bronchi

**Simple Tumours** These include adenoma papilloma lipoma myxoma fibroma chondroma and amyloid deposits

**Malignant Tumours** The growth is usually a primary carcinoma Secondary carcinoma may also involve the bronchi Sarcoma is rare

### Adenoma of the Bronchus

**Pathology** A small polypoid tumour arises usually in mucous glands in a main bronchus but occasionally in a tertiary bronchus It is generally smooth shiny and very vascular and the greater part of the tumour is situated in the wall of the bronchus only a small portion projecting into the lumen It was formerly mistaken for carcinoma owing to the staining peculiarities of certain of its cells and their irregular distribution in the connective tissue Metastases have occasionally been recorded which suggest the tumour may be potentially malignant

**Clinical Findings** The patient is usually an adult under the age of 40 It is more common in women Hæmoptysis of varying degree is *often the earliest symptom* In other cases dry pleurisy pleural effusion or empyema may be first noted If the tumour causes bronchial obstruction there is wheezing pulmonary collapse or bronchiectasis

**Differential Diagnosis** This includes other causes of hæmoptysis *pulmonary collapse, or bronchiectasis* *In cases complicated by* pleural effusion the underlying cause is likely to be overlooked The diagnosis is established by microscopical examination of a piece of the tumour removed through a bronchoscope

**Prognosis** *Cases diagnosed early can usually be treated successfully*

**Treatment** There is a risk of fatal hæmorrhage if removal is attempted by forceps through a bronchoscope or by diathermic coagulation Lobectomy is the usual treatment

geneous shadow due to an atelectatic area of lung and with infection this may become mottled or show cavity formation The walls of the cavity may be thickened and irregular The mediastinum may be displaced slightly to the affected side and the corresponding diaphragm raised The picture may be obscured by a pleural effusion in such instances the fluid should be aspirated and replaced by air when the growth may be revealed by X ray examination A bronchogram may then reveal a blocked bronchus Bronchoscopy This should be advised in all suspected cases of early carcinoma In about 25% of cases however the growth is not visible owing to its peripheral distribution In some cases of peripheral growth thoracotomy may be necessary to enable a specimen to be removed for microscopical examination

**Differential Diagnosis** This is difficult in the early stages A persistent dry cough or an increase of a chronic cough should not be dismissed as of no consequence and wheezing should not necessarily be attributed to asthma If the symptoms suggest an early lesion bronchoscopy should not be postponed until the development of advanced signs renders radical treatment impossible The Wassermann reaction should be determined to eliminate the possibility of a gumma The satisfactory response to antisyphilitic treatment in cases which give a positive reaction will help to confirm the diagnosis of a gumma I have seen such a case The presence of tubercle bacilli in the sputum does not negative the possibility of bronchial carcinoma as tuberculosis and carcinoma may coexist This also applies to the presence of a lung abscess A mesothelial mediastinal cyst—the so called spring water cyst—seen on X ray examination as a shadow in the right cardio phrenic angle may be mistaken for a bronchial carcinoma The chief symptoms may be distal due to secondary deposits as described above or due to polyneuritis Glycosuria may be discovered on routine examination caused by secondary deposits in the pancreas The irregular fever may suggest malignant endocarditis or Hodgkin s disease

**Course and Complications** The course is steadily progressive Secondary deposits are liable to occur in glands the liver the kidneys the suprarenals the brain the eye (the fundi should always be examined) the spinal cord and in bones Complications include bronchitis bronchi ectasis lung abscess and gangrene pleural effusions and hæmoptysis which may be fatal The growth may directly invade the pericardium and auricle of the heart resulting in cardiac arrhythmia

**Prognosis** Death usually occurs in 8 to 22 months from the onset of symptoms

**Treatment** In early cases in which there are no secondary deposits the whole lung may be removed by dissection pneumonectomy Pro mising results have been obtained the patient in about 25% of cases being alive 5 years after the operation An early tumour in the lower lobe can at times be removed by lobectomy Contra indications to pneumonectomy are heart disease distant metastases enlarged media stinal glands recurrent laryngeal nerve palsy Horner s syndrome

deposits may also cause brachial or sciatic neuritis 2 *Osseous* Pains in the bones or spontaneous fractures occur in about 30% of cases 3 *Gastro intestinal* Metastases in glands or in the liver may result in dysphagia jaundice, hæmatemesis or melæna 4 *Lympho glandular* Enlargement of the supra clavicular, axillary or cervical glands may simulate Hodgkin s disease

Peripheral neuritic symptoms, swelling of the joints simulating rheumatoid arthritis, and pulmonary osteo arthropathy may appear apart from secondary deposits

*The Pancoast tumour* is the name applied to an apical or superior pulmonary sulcus carcinoma The characteristic features are pain in the region of the scapula and inner side of the arm with wasting of the small muscles of the hand *There is also homolateral cervical* sympathetic paralysis (Horner s syndrome) Dulness is found at the apex of the lung and X ray examination shows a small circumscribed apical opacity with destruction of the posterior part of the first three ribs

*On Examination* In the early stage the patient often looks well and no physical signs can usually be found, and in the main stem type before any pulmonary collapse has occurred X ray examination of the chest is usually negative Clubbing of the fingers may rapidly develop With a peripherally situated growth X rays may reveal a shadow due to a small area of collapsed lung With the main stem bronchus type the physical signs which develop vary with the degree of obstruction present With partial obstruction there is slight dulness and weak air entry over the affected part of the lung With a ball valve obstruction the percussion note becomes boxy from over distention of the lung and expiration is prolonged and wheezy With complete obstruction the affected part collapses with absent breath sounds and possibly displacement of the mediastinum to the affected side In the peripheral type the signs may suggest infiltration consolidation or *abscess formation or they may be those of dry pleurisy or of pleural* effusion In the latter case exploration may reveal a serous hæmor rhagic or purulent effusion With the central type the physical signs in the lungs may be very slight over a root area posteriorly Pressure may cause collapse often of a lower lobe bronchus and further evidence of the mediastinal syndrome may be revealed (see p 209) The barium swallow may show deviation of the œsophagus indicating not only mediastinal glandular involvement but also inoperability Later the signs of infection of bronchi or of spread of the growth in the lung or of metastases may be found The mass of growth may extend so that it is palpable above the clavicle or it may ulcerate through the chest wall The sputum This may be scanty and tenacious or it may be red resembling red currant jelly or darker like 'prune juice' *Carcinoma cells have been found in the sputum in about 60%* of cases by some observers The blood In the later stages there is a microcytic anæmia Leucocytosis is not the rule unless there is secondary infection X ray examination The shadow of the growth is only revealed in a minority of cases There is more often a homo

aureus and the Klebsiella pneumoniæ (Friedlander s bacillus) Over 90% of cases of pneumonia are due to the pneumococcus and it gives rise to the most typical clinical picture

## Pneumococcal Pneumonia

**Etiology** Over 75 types of pneumococci have been described but as serum treatment has now been abandoned the actual type present in an individual case is of little moment *Predisposing causes* 1 The presence of pneumococci in the naso pharynx Probably over 50% of normal individuals are carriers of pneumococci 2 Age Children young adults and old people 3 Sex Males predominate 4 Season Autumn and winter 5 A previous attack This predisposes to subsequent attacks 6 Debility Due to exposure overwork and alcoholism

**Pathology** Pneumococcal pneumonia usually affects one or more lobes of the lung Frequently however there are scattered areas of consolidation constituting what was known as bronchopneumonia The affected lobe of the lung goes through characteristic changes In the first stage there is active hyperæmia this is followed by consolidation—red hepatisation The third stage is known as grey hepatisation and this is followed by resolution It is rare for an abscess or gangrene of the lung to develop Dry pleurisy is always found over the affected lung

**Incubation Period** This is probably from 1 to 2 days

**Clinical Findings** The patient is often an adult male of rather a robust type There may be a history of exposure to cold or wet but rarely of contact with another patient suffering from lobar pneumonia He is suddenly taken ill with shivering malaise and stabbing pain in the chest The pain may be referred to the abdomen or to the tip of the shoulder if there is diaphragmatic pleurisy A short dry and painful cough usually rapidly develops In children the onset is often with a rigor or with vomiting

*On Examination* The patient looks ill with a somewhat anxious expression dry skin flushed face and bright eyes The breathing is shallow and rapid and a grunting noise may be heard with expiration the alæ nasi muscles may be seen in action The temperature is usually high 102 or 104 F the pulse frequent 110 to 120 and the respiration rapid 30 to 40 The pulse respiration ratio may be 3 1 or even 2 1 The chest There is diminished expansion on the affected side the percussion note may be slightly impaired over one lobe the air entry there is weak and a few fine rales or pleural crepitations may be heard X ray examination at this stage may reveal some deep seated consolidation which spreads outwards later Usually on the second or third day definite signs of consolidation are found in the affected lobe These signs are dulness increased tactile fremitus tubular breathing and fine indux' crepitations The percussion note at the apex of the lung above the affected lobe may be skodaic The urine may contain a trace of albumin with diminution or absence of chlorides A small amount of tenacious blood stained

and a hæmorrhagic pleural effusion Diaphragmatic paralysis is usually a contra indication an inflammatory pleural effusion which is not blood stained is not necessarily so as it may be due to infection An exploratory thoracotomy is indicated in some cases in which the diagnosis or the operability is in doubt Deep X ray treatment offers no hope of cure Treatment in advanced cases is palliative, by sedatives such as diamorphin hydrochlor gr 1/8 as required, for relief of cough and pain or by pethidine hydrochlor Physeptone or Heptalgin (see p 43) Dyspnœa due to a pleural effusion may be relieved by aspiration

### Injury to the Bronchi

Rupture of a bronchus may result from severe external trauma to the chest wall such as in a crushing accident This is rapidly followed by surgical emphysema of the chest and neck and death usually ensues in a short time

## THE LUNGS

### Congenital Agenesis of a Lung

Rarely one lung is congenitally absent the other lung showing compensatory hypertrophy The chest appears symmetrical and the movement is equal on both sides but there is considerable displacement and rotation of the mediastinum The trachea and heart are markedly displaced the breath sounds being audible over the whole chest Provided the patient survives the first year there appears to be no reason why the absence of the lung should cause death

Other congenital abnormalities may be associated with the absence of a lung such as ectopia of a kidney a unicornate right uterus, an abnormally shaped skull hypertrophy of an adrenal imperforate anus etc

### Sequestration of a Lung

This is a congenital abnormality in which one or more segments of *a lung are separated from the surrounding lung tissue The affected* portion of the lung has a rudimentary structure the tissue often being undifferentiated It may be solid or cystic The sequestrated portion *is usually supplied by an anomalous artery arising* either from the aorta or the cœliac axis The patient may complain of pain in the back and on X ray examination a shadow is seen which may be mistaken for that cast by a neoplasm or atypical pneumonia An aortogram may demonstrate the anomalous artery and so confirm the diagnosis

### The Bacterial Pneumonias

Pneumonia and influenza are still important causes of death especially in the aged and in infants Pneumonia whenever possible should be classified etiologically rather than anatomically and for this reason there is a tendency to discard the older divisions into lobar and bronchopneumonia The most important causes of pulmonary inflammation and pneumonia are the streptococcus (Diplococcus) pneumoniæ or pneumococcus the streptococcus hæmolyticus the *staphylococcus*

injections are begun on the first day when the signs are slight the temperature pulse and respiration rates will often fall to normal within 48 hours The general condition of the patient rapidly improves pain disappears and toxic symptoms are abolished On the second or third day however the physical signs of consolidation may be marked despite the absence of all toxic signs The following are the most important complications Delayed resolution pleural effusion (serous or purulent) an empyema may develop during the acute stage of the pneumonia (syn pneumonic) or after the temperature has fallen to normal (meta pneumonic) pericarditis (dry serous or purulent) otitis media endocarditis meningitis venous thrombosis nephritis colitis peritonitis jaundice arthritis parotitis peripheral neuritis abscess or gangrene of the lung Fibrosis of the lung and bronchiectasis may occur as sequelæ

**Prognosis** This is usually good with the administration of anti biotics The prognosis of staphylococcal and Friedlander s pneumonias is much more grave than is that of pneumococcal pneumonia The following are unfavourable factors The extremes of age a history of chronic alcoholism coexisting diseases such as heart lesions nephritis or diabetes mellitus pregnancy a severe degree of toxæmia a low degree of fever and absence of response ( asthenic type) as shown by a leucocyte count below 10 000 per c mm Double pneumonia or creeping pneu monia is more serious than when the disease remains confined to one lobe Unfavourable signs are a falling blood pressure with rising pulse rate progressive weakening of the heart with dilatation marked cyanosis and a respiration rate of over 50 a minute Death usually occurs from vasomotor collapse with pneumococcal bacteriæmia

## Streptococcal Pneumonia

**Etiology** The streptococcus is $\beta$ hæmolytic group A The disease is often secondary to acute infections especially influenza and measles or to hæmolytic streptococcal tonsillitis

**Pathology** The changes in the lungs are often widely scattered assuming a bronchopneumonic distribution

**Clinical Findings** The onset is usually insidious with bronchitis If untreated the disease tends to progress rapidly the patient being prostrated and toxæmic Pleural effusion frequently occurs Clinical examination reveals patchy areas of consolidation with coarse rales

**Complications** Streptococcal septicæmia and empyema are likely to occur unless the disease is treated early and efficiently

## Staphylococcal Pneumonia

**Etiology** The causative organism is the staphylococcus aureus type A hæmolytic and coagulase positive

**Pathology** There is a great tendency to abscess formation Empyema and broncho pleural fistula are not uncommon

**Clinical Findings** In adults the disease is often secondary to staphylococcal infection elsewhere such as perinephric abscess or boils but in many cases the primary focus cannot be found It is also met

('rusty') sputum is usually brought up on the second or third day and the sputum continues during the course of the illness, but gradually becomes looser and free from blood. In other cases there may be a definite hæmoptysis with bright blood. Labial herpes is often seen during the early stages of the illness. The blood pressure is generally low, and the blood count shows a leucocytosis of 20 000 to 30 000 per c mm in "sthenic" types with a good reaction.

The use of antibiotics has considerably modified the classical course of the disease. The classical picture is as follows. The temperature remains raised for 5 or 6 days and then may fall to normal rapidly by crisis or more gradually by lysis. At the crisis the patient's condition generally improves, there is sweating and the toxæmic symptoms usually abate but the patient remains exhausted and collapse may occur with signs of heart failure. The physical signs of consolidation remain unchanged.

Shortly after the crisis the patient may become maniacal but this does not usually persist for more than a day or so. As resolution occurs in the lungs there is an increase of the crepitations heard ('redux' crepitations) and the signs of consolidation gradually disappear.

**Differential Diagnosis.** The diagnosis of lobar pneumonia is often a matter of no difficulty. In the early stages it may be confused with the onset of various acute illnesses especially influenza and enteric fever. The acute pain in the side suggests pleurisy, and this is almost invariably present in pneumonia. The initial fever and vomiting may suggest the onset of scarlet fever in children but the diagnosis is soon established in the latter case by the appearance of the characteristic rash. Apical pneumonia may be mistaken for meningitis. The onset and early stages of acute pneumonic tuberculosis and of lobar pneumonia are very similar. In the former the temperature does not fall rapidly with treatment as it does in pneumococcal pneumonia and tubercle bacilli are usually found in the sputum. When the pain in pneumonia is abdominal it may suggest an acute abdominal lesion such as appendicitis. The increase in the respiration rate in pneumonia is an important diagnostic sign. Massive collapse of the lungs was formerly usually mistaken for post operative pneumonia but the displacement of the heart to the affected side in massive collapse and the course of the disease serve to differentiate the two. In paroxysmal tachycardia acute congestion may be found at the base of a lung suggesting early pneumonia but a careful enquiry into the history of the illness and clinical examination establish the diagnosis. Early maniacal symptoms in lobar pneumonia may lead to an error in diagnosis. Primary atypical pneumonia may give rise to difficulty in diagnosis in some cases. In this condition headache is usually more severe and respiratory symptoms are less marked. Examination of the sputum shows the causative organism in bacterial pneumonias and examination of the blood reveals the presence of cold agglutinins in primary atypical pneumonia.

**Course and Complications.** The classical stages are considerably modified by the early administration of penicillin and other antibiotics and to a less extent by the use of sulphonamides. If the penicillin

Cascara Evacuant m 30 to 60 or an enema may be given if necessary Cataplasma kaolini may be applied to the chest on lint over the affected portion of the lung and changed every 12 hours A light gamgee jacket should be worn over this If the pain in the chest is severe it can usually be rapidly relieved by strapping the affected side of the chest in a position of full expiration Sleep should be secured by giving pulv ipecac et opii gr 10 the first two evenings and later by the use of two Soneryl tablets gr 1½ each In some cases of obstinate insomnia brandy fl oz 1 in hot water at night acts like a charm If the patient is an alcoholic whisky or brandy fl oz ½ should be given four to six hourly For the irritating cough a sedative linctus may be prescribed such as syrup codein phosphat (B P C) m 60 occasionally Later as expectoration loosens a stimulating mixture should be given such as Ammon carb gr 5 pot iod gr 3 tnc scillæ m 20 sp chlorof m 7 infus senegæ rec ad fl oz 1 Fl oz 1 t d s If there is cyanosis oxygen should be given continuously through a B L B mask or a polymask An oxygen tent is of great value for babies Signs of cardiac dilatation or weakening of the pulse should be treated by the administration of such drugs as Coramine 1 5 ml subcutaneously four hourly or liq adrenal hydrochlor m 5 subcutaneously six hourly Digitalis in pneumonia is only required for congestive failure or for auricular fibrillation or flutter Flatulent abdominal distention must be treated early as it embarrasses both heart and lungs For this purpose a rectal tube should be passed a turpentine enema given containing turpentine fl oz 1 in 15 fl oz of starch mucilage or turpentine stupes applied to the abdomen prepared by sprinkling m 60 of turpentine over a hot fomentation If these measures fail a hypodermic injection should be given of 1 ml of Pituitrin For delirium and restlessness an ice compress should be applied to the head and a rectal injection given of pot brom gr 120 in water fl oz 2

During convalescence breathing exercises must be carried out to expand the lungs These should be inspiratory in nature and not expiratory as in blowing through Wolff s bottles By this means the risk of subsequent bronchiectasis is much diminished Resolution should be checked by X ray examination

**Hæmolytic Streptococcal Pneumonia** The hæmolytic streptococci are very sensitive to penicillin which should be given in large doses such as 1 million units of crystalline penicillin 2 or 3 times in the 24 hours

**Staphylococcal Pneumonia** Penicillin will have to be given in large doses for many weeks to prevent a relapse or recurrence of the disease One million units of crystalline penicillin should be injected intramuscularly every 12 hours for 2 or 3 months In very severe cases as much as 15 or 20 million units every 24 hours are required If the staphylococci are shown to be resistant to penicillin aureomycin or Achromycin should be given according to the sensitivity tests The dose is 2 capsules (250 mg each) by mouth every 6 hours for 3 or 4 weeks Beplex capsules 2 t d s should also be given

**Friedlander s Pneumonia** Streptomycin sulphate should be injected

with in infants and, if the child survives may lead to bronchiectasis or *cystic changes in the lungs* The onset is insidious, but the disease tends to progress rapidly High irregular fever with profuse sweating are characteristic features When an abscess bursts into a bronchus pus is expectorated There is a tendency for the abscesses to leave thin walled cavities as shown by X ray examination

### Friedlander's Pneumonia

**Etiology** This variety of pneumonia is due to infection of the lungs with Friedlander s bacillus

**Pathology** There are numerous small areas of consolidation in the lungs which tend to coalesce They form small pulmonary abscesses

**Clinical Findings** The patient is often over middle age, and may be debilitated or a chronic alcoholic The onset is usually acute with fever shivering attacks and pains in the chest due to pleurisy There is copious purulent sputum The disease rapidly progresses to a fatal issue if the appropriate antibiotic treatment is not given On X ray examination it is found that the disease tends to affect the upper lobe with cavity formation and in this respect it resembles pulmonary tuberculosis

**Prognosis** The mortality rate has been lowered from about 80% to about 20% with modern treatment

### Treatment

**Pneumococcal Pneumonia** The patient should retire to bed at the first symptom It is not possible at the first visit to know the nature of the causative organism There may be no sputum for 2 or 3 days and *there may be no facilities for isolating the organism from the sputum* and for determining its antibiotic sensitivity in every case there will be a further delay before the result is obtained Treatment however *should begin as soon as the patient is seen As in the vast majority* of cases of bacterial pneumonia the causative organism is the pneumo coccus crystalline penicillin should be injected intramuscularly *500 000 units every 6 hours for 24 hours then 500 000 units every* 8 hours for 24 hours and then 500 000 units every 12 hours for 24 hours Procaine penicillin may now be injected intramuscularly 600 000 units *daily for about another 4 days Alternatively the patient may* be given a sulphonamide such as sulphadimidine the initial dose being 2 G followed by 1 G every 6 hours for 5 or 7 days A tetracycline *drug can also be used either aureomycin or Achromycin* The dose is mg 500 (2 capsules) every 6 hours for 5 to 7 days Should diarrhœa occur with these drugs they should be discontinued Vitamin B should be given *in the form of Beflex capsules 2 t d s*

*The diet* A milk diet should be given during the acute stage The patient should drink plenty of fluid such as dextrose orangeade (dextrose oz 4 *the juice of an orange* and water a pint) and barley water The diet can usually be rapidly augmented by thin bread and butter and a lightly boiled egg and steamed fish The bowels should be opened daily or *every other day* by a saline aperient by cascara sagrada gr 2 or by

culous bronchopneumonia is not often diagnosed in a child before death although the course tends to be more prolonged and tubercle bacilli may be found in the fæces from swallowed sputum

**Treatment** If the patient is an infant he must be in bed but should be taken in the arms from time to time to change his position The shoulders and head should be slightly raised The temperature of the room should be kept at 65 F day and night, and the air moistened with a steam kettle especially in cases of obstructive dyspnœa If the temperature rises over 105 F or there is much restlessness the infant should be tepid sponged with water at 85 to 92 F The diet consists of 6 to 8 oz of equal parts of milk and water containing 2 teaspoonfuls of lactose every 3 hours This is given by a spoon or by bottle If there is curd indigestion the feeds are citrated using sod citrat gr 2 to each ounce of undiluted milk Drinks of half strength normal saline (0 42% sod chlorid ) are given between feeds and if the infant is dehydrated a continuous subcutaneous drip of normal saline is also advisable The bowels should be opened every 2 or 3 days by syrup ficorum m 60 nocte or a glycerin suppository may be used Cyanosis should be relieved by the use of an oxygen tent Achromycin may be given by mouth the total daily dose according to weight being 50 mg /8 lb 100 mg /16 lb 150 mg /2 stones 250 mg /3 stones and 500 mg /6 stones Pædriatic drops contain 5 mg /drop the syrup 125 mg /teaspoonful the oral suspension 250 mg /teaspoonful and the tablets or capsules 250 mg each Alternatively for a severely ill child aged 1 year 300 000 units of a procaine penicillin may be injected every 12 hours For circulatory failure Coramine 0 5 ml should be injected subcutaneously every 4 hours For restlessness and insomnia brandy m 20 in ½ fl oz of water may be given and repeated in 4 hours if necessary

## The Viral and Rickettsial Pneumonias

Virus infections causing pneumonic changes in the lungs include those due to measles small pox influenza and psittacosis Primary atypical pneumonia is thought to be due to a virus which has not yet been discovered Rickettsial pneumonia is exemplified by Q fever

## Primary Atypical Pneumonia

(*Virus pneumonia Virus pneumonitis*)

**Definition** An acute catarrhal condition of the lungs usually benign in character probably caused by a virus and characterised radiologically by soft shadows in the lungs localised to one or more bronchopulmonary segments

**Etiology** The virus has not been isolated A non hæmolytic streptococcus M G has been obtained from the lungs and specific antibodies have been found in the blood of about 50% of patients during convalescence It has not been transmitted to animals but human volunteers have been infected by spraying the nose and throat with a bacterium free filtrate obtained from throat washings and sputum of patients suffering from the disease

intramuscularly first 1 G every 8 hours for 24 hours this is reduced to 1 G *every 12 hours for 48 hours*, and then to 1 G daily until the temperature has been normal for a week Sulphadimidine should be simultaneously administered by mouth an initial dose of 2 G then 1 G every 6 hours for a week, and then 1 G t i d for 2 or 3 weeks

### Other Bacterial Pneumonias

These include infections by the H influenzæ the B anthracis the P tularensis the P pestis the Mycobacterium tuberculosis, the H pertussis and the Bact typhosum

The H influenzæ is sensitive to streptomycin which may be used in combination with sulphadimidine

### Bronchopneumonia in Infants

(*Capillary bronchitis*)

**Etiology** This is a disease of infants and young children, due to a mixed infection of the terminal bronchioles with such organisms as the H influenzæ the staphylococcus Friedlander s bacillus the pneumococcus and the streptococcus It may also occur as a complication of such diseases as measles and whooping cough

**Clinical Findings** The patient is usually an infant or young child The onset is comparatively sudden with the symptoms of a feverish cold

*On Examination* Inspection The child looks flushed or cyanosed restless and ill The respirations are rapid expiration grunting and a dry cough may be heard The alæ nasi muscles are seen to be in action *and there may be recession of the intercostal spaces and retraction of the* xiphisternum with inspiration Palpation Rhonchal fremitus may be felt, if there is an associated large tube bronchitis Percussion Small *areas of impaired resonance may be detected especially in the lower lobes* behind Auscultation The air entry is often weak over the lower lobes and harsh over the upper lobes Small patches of bronchial breathing *may be heard with bronchophony and a few fine crepitations* Often however no sign of consolidation can be detected but rales may be heard over the lower lobes *In confluent bronchopneumonia larger areas of consolidation are present The heart sounds are rapid and the pulmonary* second sound is usually accentuated The temperature rises rapidly to 103 or 105 F and then becomes remittent in type falling by crisis or *lysis in about 7 to 10 days A further rise in temperature may indicate* a fresh spread of the infection The respirations may rise to 40 or 60 or even higher in a severe case and the pulse rate to 120 or more Infants *have no sputum any pulmonary secretion which is brought up on* coughing being either swallowed or vomited In severe cases the child is very drowsy being overwhelmed with the toxæmia

***Differential Diagnosis*** There is usually little difficulty in the diagnosis but the extreme dyspnœa may suggest laryngeal diphtheria There is however no obstruction to the airway through the larynx *In some cases the cerebral symptoms may suggest meningitis* Tuber

**Prognosis** Antibiotic treatment has shortened the duration of the illness Recovery is the rule but the X ray shadows may not disappear for some weeks after convalescence is firmly established The mortality rate is about 0 2% and encephalitis is a dread complication

**Treatment** The patient should be kept in bed until the temperature has been normal for a week and he should not return to work until the sedimentation rate of the red cells is normal and the X ray shadows have disappeared Aureomycin or terramycin should be administered by mouth in doses of 500 mg every 6 hours for 5 to 7 days A dramatic response is often obtained Steam inhalations may relieve the distressing cough For severe cyanosis and dyspnœa oxygen should be administered by nasal catheter or the B L B mask

## Pneumonitis

There is a localised area of inflammatory exudation in the lungs This may be due to 1 Chemical irritants These include irritant gases such as ammonia phosgene and mustard gas Manganese pneumonitis may occur in men employed in the manufacture of potassium permanganate and beryllium pneumonitis in fluorescent bulb workers 2 Deep X ray treatment Excessive irradiation as in the treatment of carcinoma of the breast may cause pneumonitis and later fibrosis of the lung This may be very severe causing intense dyspnœa and mediastinal displacement 3 Inhalation of oil Lipid pneumonitis is due to droplets of mineral or cod liver oil especially when forcibly administered to debilitated infants It may also result from the use of nose drops containing mineral oil or from taking liquid paraffin at night in patients suffering from dysphagia or even from constipation Paraffin should never be taken at night

## Aspiration Lung Infections

Under this heading are included patchy areas of consolidation which may result from inhalation of septic material from the naso pharynx They may therefore be secondary to infections in the nose and throat to inhalation of vomit or of a foreign body Septic material may be coughed from the diseased to the healthy lung as in bronchiectasis In chronic bronchitis infected mucus may block a bronchus and cause an area of collapse with high temperature the condition rapidly clearing when the mucus is expectorated This is not uncommon in children Aspiration pneumonia is also met with in association with bronchial carcinoma In the more severe forms of aspiration pneumonia localised areas of suppuration form which may give rise to chronic lung abscess

## Pulmonary Tuberculosis

**Definition** Tuberculous infection of the lungs bronchi bronchial glands or pleuræ

**Etiology** Pulmonary tuberculosis is caused by the Mycobacterium tuberculosis Both human and bovine types affect man The organisms

**Pathology** The affected alveoli are filled with coagulated serum There are localised areas of pulmonary collapse The primary lesion is thought to be a peri bronchitis and peri bronchiolitis Encephalitis is found in some cases

**Clinical Findings** The disease often occurs in small epidemics The incubation period varies from 2 to 21 days or longer Frequently there are no catarrhal signs at the onset but there may be a dry, racking cough with headache and limb pains Some cases are apyrexial, but the temperature usually rises to 100° F or 103° F and after remaining raised for 5 to 10 days falls to normal by lysis The sputum is mucoid never rusty ' or " prune juice in colour but small hæmoptyses of a teaspoonful or so may be met with It contains no characteristic or predominating organisms The physical signs in the lungs are variable, small areas of collapse indicated by slight dulness and weak air entry may appear from day to day The most characteristic sign is the presence of showers of rales heard after cough over the affected areas The pulse rate is often slower than would be expected from the temperature In severe cases there is cyanosis and marked prostration The white cell count is usually normal but there may be a slight leucopenia The sedimentation rate of the red cells is increased to between 20 and 40 mm % at one hour The blood Wassermann reaction may be positive for a short time during the acute phase of the illness and cold agglutinins are often found in the serum in high titre from the second to fourth weeks of the illness using varying dilutions of the patient s serum and a washed suspension of group O human erythrocytes The radiological findings are often more extensive than would be expected from clinical examination of the chest There may be patchy opacities somewhat resembling those seen in bronchopneumonia They are often irregular in shape and vague in outline with a hazy, ground glass appearance Over 80% of the lesions are situated at the base and owing to areas of atelectasis the diaphragm may be raised on the affected side In other cases the lesion is apical, or a lateral film may show the shadows situated in the apex of the lower lobe

**Course and Complications** In the average case the temperature falls to normal in 7 to 10 days but secondary rises of temperature sometimes occur Complications include dry pleurisy encephalitis venous thrombosis in the legs polyarthritis fractured ribs from coughing and rarely a pleural effusion An acute hæmolytic anæmia and hæmoglobinuria may be due to the cold agglutinins

**Differential Diagnosis** Apical lesions may be mistaken for pulmonary tuberculosis or bronchial carcinoma An area of basal bronchiectasis with surrounding pneumonitis may give rise to difficulty The absence of early catarrhal symptoms is opposed to a diagnosis of influenza or tracheobronchitis Failure to respond to penicillin usually excludes the diagnosis of lobar pneumonia At times a prolonged fever with a relatively slow pulse rate and low white cell count suggest the diagnosis of enteric fever In some cases it may be necessary to exclude pneumonic consolidation due to psittacosis Rickettsia infections such as the Q fever of Australia American Q fever and ornithosis

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## Pulmonary Tuberculosis

**Definition** Tuberculous infection of the lungs bronchi bronchial glands or pleuræ

**Etiology** Pulmonary tuberculosis is caused by the Mycobacterium tuberculosis Both human and bovine types affect man The organisms

may infect the body from dust or dried sputum by droplet infection or by contaminated articles of food such as milk, cream butter cheese or meat Contact with 'open or sputum positive cases is probably the most important source of infection The incidence of tuberculous infection can be determined by the Mantoux test (see p 165) by which it has been shown that infection from birth to 5 years is five times greater in children who are contacts with sputum positive cases than in those who are non contacts Further between the ages of 0 to 15 years contacts are infected twice as often as non contacts This is illustrated in the chart (see Fig 11) Close contact with open cases of pulmonary tuberculosis as in households or at work is a very important cause of active tuberculosis in adults Conjugal infection is a very real danger which has been under estimated in the past *Predisposing causes* 1 *Heredity* *The disease undoubtedly occurs in families and in some* rare instances infants may be born with tuberculosis 2 Race Virgin races are very susceptible *i e* those which have not previously suffered from the disease and which as the result of civilisation are brought in contact with it 3 Sex There is a slight predominance of males 4 Age The maximum age incidence of pulmonary tuberculosis is between 15 and 45 years 5 Climate Pulmonary tuberculosis is prevalent in districts exposed to rain bearing winds 6 Sanitation Overcrowding and absence of sunlight and fresh air are potent predisposing factors 7 Occupation Inhalation of dust especially silica particles Tin miners are prone to the disease whereas coal miners are relatively immune 8 General health Lack of food overwork chronic alcoholism, diabetes mellitus and measles predispose to tuberculosis

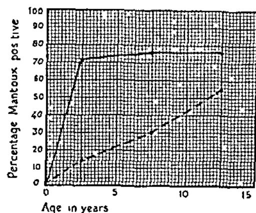


FIG 11 MANTOUX SKIN TESTS DOW AND LLOYD *Brit Med Journ* WINGFIELD *The Practitioner*

**Pathology** The tubercle bacilli may reach the lungs by three routes By the respiratory tract by the blood stream and by lymphatics In childhood infection is chiefly from the alimentary tract due to infected milk The organisms are of the bovine type and pass by lymphatics to the bronchial glands and thence to the lungs The bacilli may also travel from the tonsils to the cervical glands and thus to the lungs Pathologically there are two types of pulmonary tuberculosis exudative and productive Exudative changes occur when tubercle bacilli reach the alveoli The alveoli are filled with an exudate containing mononuclear cells and the condition is tuberculous pneumonia In productive tuberculosis granulation tissue forms which constitutes the tubercle This may disappear or it may caseate and

form a cavity The solid solitary lesion seen on X ray films known as a coin shadow or tuberculoma is due to liquid or caseous material in a cavity whose bronchus has become blocked

Dissemination of tubercle bacilli from a caseous focus in the lungs may be bronchogenic hæmatogenous or lymphatic Bronchogenic spread leads to lesions in the same or opposite lung hæmatogenous dissemination to tuberculosis in distant organs such as the bones kidneys joints or to miliary tuberculosis and lymphatic spread results in tuberculous lesions in the root and supraclavicular glands

**Types of Pulmonary Tuberculosis** *Primary pulmonary tuberculosis in children* The primary focus is small It may occur in any part of the lungs often in a lower lobe and it constitutes the Ghon s focus The corresponding hilar glands are rapidly infected and enlarge These two lesions constitute *the primary complex* They caseate and heal by calcification The root lesion is larger than the primary focus The infected bronchial glands may compress a bronchus and cause epi tuberculosis This is either tuberculous pneumonia or atelectasis of a pulmonary segment

Less often healing does not occur and a *progressive primary complex* develops which may be fatal The changes produced in the lungs resemble those in adult tuberculosis and bronchogenic or hæmatogenous spread may occur

*Primary pulmonary tuberculosis in adults* When a Mantoux negative young adult such as a medical student or nurse is found to have become Mantoux positive what is known as Mantoux conversion has taken place This means that he has developed a tuberculous lesion usually in the lungs as the result of contact infection at his work Often the individual does not feel ill and no X ray changes are seen In other cases months later a small focus is seen nearly at the apex of a lung (Assmann s focus) This may be progressive and lead to cavita tion but usually it heals by fibrosis and not by calcification as does the primary infection in children The reason for this is not known

*Re infection pulmonary tuberculosis in adults* This may be due to a reactivation of a childhood infection in the lungs or elsewhere but more often it results from a recent infection by inhalation from contact with a person suffering from pulmonary tuberculosis In childhood infections the organism is almost always of the bovine variety in adult tuberculosis the organism in the vast majority of cases is of the human type

Pathologically there is a blending of exudative and productive lesions Caseation and cavitation occur localised healing and localised extension take place simultaneously and fibrotic changes ensue but there is little tendency to calcification Clinically this type of tuber culosis includes the majority of cases met with in young people and adults but many cases we see in adult life are first infections no child hood infection having taken place Bronchogenic spread is common hæmatogenous spread is rare and the root glands are seldom enlarged

*Tracheobronchial tuberculosis* It was only with the more extensive use of bronchoscopy in pulmonary tuberculosis that bronchial tuber culosis was recognised It is important as it may cause ulcerative

lesions in the larger bronchi with subsequent stenosis which prevents the closure of lung cavities, and necessitates pulmonary resection rather than collapse therapy

Clinical Findings Pulmonary tuberculosis may have an insidious or acute onset The former is the more common *The insidious onset* Mass miniature radiography has shown that the initial lesion is often symptomless The early symptoms are very numerous amongst the most important are the following Cough which persists for several months usually with expectoration Lassitude and loss of weight *Palpitations or dyspnœa on exertion Nervous debility which may* simulate neurasthenia Amenorrhœa is not infrequent In other cases hoarseness is the earliest symptom Some patients complain of period ical shivering or sweating The symptoms may date back to what the patient designates an attack of influenza, or to a cold which has never properly disappeared In children an attack of measles or whooping cough may be followed by persistent ill health leading up to the diagnosis of tuberculosis *The acute onset* This may also be of several varieties Hæmoptysis The patient is apparently in good health and suddenly coughs up blood in varying amounts Spontaneous pneumothorax Here also the patient is apparently well, when the pneumothorax suddenly occurs with severe pain and dyspnœa Pleurisy This may be acute dry pleurisy or pleurisy with effusion Acute influenzal type Acute *pneumonic or bronchopneumonic tuberculosis ("galloping con* sumption) The onset closely resembles that of lobar or broncho pneumonia Acute miliary tuberculosis The patient is suddenly taken ill with a high fever malaise and dyspnœa This is more common in children than in adults

*On Examination* 1 *Chronic pulmonary tuberculosis* The early stages There is no characteristic appearance and the patient is not usually wasted although the weight is often below his highest known weight *Examination of the chest* Inspection There may be drooping *of one shoulder with slight flattening and diminished expansion of* the upper part of the chest on the affected side Palpation Tactile fremitus is slightly increased over this area Percussion The note is slightly impaired at the upper part of the chest and Kronig s area of apical resonance above the clavicle may be diminished Auscul tation The breath sounds over the affected area of lung may be weal expiration may be prolonged or the air entry jerky The breath sounds in other cases are broncho vesicular or vesiculo bronchial At the back the breath sounds in the supraspinous region may be coarse and rough *(Grancher s granular breathing) The air entry may be* weaker at the base on the affected side Often there are no adventitious sounds or an occasional rale may be heard after cough There is usually slight bronchophony and faint pectoriloquy The special signs and symptoms indicative of activity are described below

*Consolidation of the Lung* The signs in the chest are more marked The note is dull tactile fremitus is increased the breath sounds are bronchial usually some coarse rales are heard and there is bronchophony and pectoriloquy

*Caseation of the Lung* There is dulness with increased tactile fremitus the breath sounds are hollow and bronchial and after cough showers of crackling and bubbling rales are heard There is also bronchophony and pectoriloquy

*Excavation of the Lung* There is flattening of the chest wall over the cavity the note is hyperresonant over the cavity if it contains air and if it is in communication with a bronchus a cracked pot sound may be heard on percussion (*bruit de pot felé*) The breath sounds are amphoric or cavernous and coarse or metallic râles are heard There is intense bronchophony and pectoriloquy and immediately after cough a hissing sound may be heard (post tussive suction) When a large cavity contains air and fluid a coin sound and ægophony may be present If the cavity is filled with fluid the physical signs are dulness weak breath sounds and diminished voice conduction

*Fibrosis of the Lung* There is flattening of the chest wall with diminished tactile fremitus dulness weak air entry and coarse leathery or sticky rales Vocal resonance is diminished If the fibrosis affects the left upper lobe pulsation may be seen in the second and third left spaces close to the sternum caused by retraction of the lung and uncovering of the heart The trachea may be displaced to one or other side and this may occur without any cardiac displacement if the fibrosis affects the upper part of the mediastinum only

Various lesions are often combined in chronic pulmonary tuberculosis such as caseation or consolidation with fibrosis

2 *Chronic miliary tuberculosis* The patient is usually between the ages of 11 and 30 years and may complain of cough dyspnœa pain in the chest expectoration and at times of hæmoptysis The evening temperature is usually a little raised The signs in the lungs are very slight a few scattered râles being heard at several points The spleen may be palpable and glands bones or joints may show tuberculous lesions Diagnosis can be established in many cases by finding tubercle bacilli in the sputum stomach washings or in a pleural effusion if it is present X ray examination shows small rather soft nodular shadows scattered throughout the lung fields These were formerly diagnosed radiologically as being due to acute miliary tuberculosis The patient however is often ambulant and not very ill the course of the disease may be prolonged for a year or more and arrest may occur The differential diagnosis includes other causes of miliary or submiliary infiltration of the lungs as seen on X ray examination such as carcinomatosis pneumokoniosis sarcoidosis bronchiolitis leukæmia kala azar polyarteritis nodosa the rheumatoid lung the lung in scleroderma bacillary dysentery bilharziasis streptococcal infiltration silicosis actinomycosis xanthomatosis pulmonary hæmosiderosis associated with mitral stenosis and tropical eosinophilia with pulmonary shadows (Weingarten s disease) and Loeffler s syndrome which is met with in children

*General Examination* The fingers are often clubbed in the early stages the nails being curved like a parrot s beak or puffin s bill Drum stick clubbing is more often associated with bronchiectasis The cheeks

ears and lips may show slight degrees of cyanosis The temperature is usually raised if there is activity present, as described later, and the pulse is also quickened The sputum This possesses no characteristic appearance It may be thin and frothy, or thick and purulent In advanced cases it is often green and nummular floating on water in flat circular masses about the size of a shilling Blood may be present in streaks or clots or the sputum may be uniformly coloured pink An examination for tubercle bacilli should be made on several occasions in every doubtful case but negative results do not exclude a diagnosis of tuberculosis The sputum may also be cultivated for tubercle bacilli In the case of children or patients with no sputum a few droplets of mucus may be coughed up and collected upon a laryngeal mirror or swab held at the back of the throat At times tubercle bacilli may thus be found The blood There are again no characteristic changes The rate of sedimentation of the red cells in citrated blood is an indication of activity in tuberculosis and a guide to progress but it is not a diagnostic test of tuberculosis Tuberculin tests are only indicative of a tuberculous infection at some time in the life of the patient, and not of present activity

*X ray Examination* This should form part of the routine examination of every suspected case of pulmonary tuberculosis although tuberculosis cannot be diagnosed solely on a film Deep seated cavitation can best be revealed by tomography

3 *Acute influenzal type* Attention has been drawn to the acute form of onset resembling influenza in which the patient is not nearly so ill as in the bronchopneumonic variety

*On Examination* Some rales are heard below the clavicle in the second and third spaces, and the X ray films show infiltration usually in the upper zone below the clavicle less often in the middle or lower zone

4 *Acute pneumonic tuberculosis* The mode of onset resembles that of lobar or lobular pneumonia but in some cases it is not quite so sudden In children acute tuberculosis often follows an attack of measles or whooping cough whereas in adults there has usually been a pulmonary tuberculous focus for some time as shown by fibrosis with mediastinal displacement

*On Examination* *Signs of consolidation are generally found* in the lower lobe of one lung The temperature is raised to 101 or 103 F and remains continuously so for a week or ten days A crisis *does not occur* but the temperature begins to swing more becoming remittent in type and the signs of toxæmia are more evident The cyanosis of a mauve tint is very characteristic with tachycardia and dyspnœa The physical signs become more those of caseation than of consolidation The diagnosis is established by finding the tubercle bacilli in the sputum

5 *Acute miliary tuberculosis* In addition to the symptoms mentioned on p 162 examination of the chest may reveal remarkably few abnormal signs or there may be râles scattered through the lungs Drenching night sweats are often a marked symptom but there are usually no rigors The temperature is of the remittent or intermittent

type and it may be inverted the morning temperature being higher than the evening X ray examination shows changes closely resembling those found in chronic miliary tuberculosis (see p 163) and both lungs may present a 'snow storm' appearance. The patient rapidly becomes more ill the spleen may be palpable and death usually occurs from profound toxæmia in 2 weeks to 8 months

*The following are the signs indicative of active disease* The temperature A rise of mouth temperature, to 99 F or over usually in the afternoon or evening This may occur when the patient is at rest or only after exercise In some cases activity may be present when the temperature does not rise above normal a subnormal swing being found The pulse Persistent rapidity at rest is usually an indication of activity The sputum The presence of tubercle bacilli X ray evidence of cavitation or of spread of the disease Other indications of active disease are an increased rate of sedimentation of the red cells loss of weight increase of cough and expectoration night sweats and lassitude

**Differential Diagnosis** The chief difficulty arises in the diagnosis of the early case Great attention should be paid to the various symptoms detailed on p 162 A temperature record should be obtained with the patient in bed and if he is afebrile the effect on the temperature of the patient getting up and exercising should be determined The lungs must be X rayed and special knowledge is required for the correct interpretation of the films The X rays may reveal a shadow due to infiltration of the lung parenchyma but this infiltration may be due to causes other than tuberculosis In *sarcoidosis* (Besnier Boeck Schaumann disease) a diffuse mottling of the lung fields may be seen on X ray examination resembling miliary tuberculosis (see p 163) The sputum should be examined repeatedly for tubercle bacilli and if not found the stomach washings and fæces should also be tested Other causes of ill health which especially require exclusion are tachycardia and loss of weight due to hyperthyroidism and pyrexia due to septic foci elsewhere In cases of doubt the Mantoux intradermal tuberculin test can be applied A positive reaction only implies that the patient has been infected with tuberculosis at some period of his life not that he has active disease now A negative reaction on the other hand is strong evidence against the diagnosis of active pulmonary tuberculosis It is performed by injecting into the skin of the forearm 0 1 ml of old tuberculin (O T ) diluted to 1/10 000 The arm is observed 48 hours later and a positive reaction consists of a central zone of œdema not less than 5 mm in diameter surrounded by a zone of erythema If the test is negative it is repeated 24 hours later using now 0 1 ml of 1/1 000 O T If this test is negative a third test is made with 0 1 ml of 1/100 O T The Vollmer s patch test and the tuberculin jelly patch test are alternative methods of applying the tuberculin test but are probably not so reliable When there are definite signs in the lungs such conditions as bronchitis and emphysema a new growth actinomycosis and pulmonary fibrosis due to other causes such as syphilis or pneumokoniosis must all be differentiated from tuberculosis The difficulty is increased

by the fact that pulmonary tuberculosis may coexist with any of these conditions

**Course and Complications** *Acute caseous tuberculosis* This usually rapidly extends and proves fatal in 2 to 3 months arrest however does occur in a certain proportion of cases *Acute miliary tuberculosis* This is often fatal in 2 weeks to 3 months *Chronic pulmonary tuberculosis* A very variable course must be expected depending largely on the resistance of the patient and the response to treatment Thus an early lesion may be almost immediately arrested by fibrosis or calcification, or it may gradually extend Periods of activity may alternate with times of comparative arrest

The following complications are of great importance Endobronchial tuberculosis This results in bronchial stenosis, and is discovered by bronchoscopy The bronchi may become infected by direct infiltration from the lungs from infected sputum by infection caused by the blood or lymph, or as part of the primary infection It may give rise to areas of pulmonary collapse to obstructive emphysema to tension cavities or to bronchiectasis If the larger bronchi are affected there may be localised wheezing A positive sputum with no evidence of active disease is always suggestive of endobronchial tuberculosis Laryngitis catarrhal or tuberculous Pleurisy, dry or with effusion Pneumo thorax Intestinal tuberculosis This results from swallowing sputum containing tubercle bacilli The lesion is usually in the ileo cæcal region X ray examination usually shows that the barium does not fill the cæcum when the ileum ascending and transverse colons are filled Fistula in ano Meningitis Bronchitis and emphysema Spontaneous subcutaneous emphysema rarely occurs Asthma Bronchiectasis Tuberculosis of the epiglottis tonsils trachea pharynx tongue or nose Tuberculosis of the epididymis prostate bladder peritoneum or kidneys Venous thrombosis Peripheral neuritis Amyloid degenera tion Rib abscess Myocarditis and pericarditis

**Prognosis** This has been much improved by antibiotic treatment Complications increase the gravity of the disease especially diabetes mellitus and tuberculous enteritis Meningitis is a very serious complica tion Freedom from financial worry enables life to be passed under the most favourable conditions and thus improves the prognosis Further the prognosis does not depend entirely upon the activity or extent of the disease but the type of disease present is of the greatest importance Thus bilateral disease with extensive fibrosis associated with thick walled cavities is always unfavourable

**Treatment** *Prophylactic* Public health authorities are concerned with prophylaxis Good housing and sanitation and a pure supply of milk are urgently needed Bovine tuberculosis in childhood could be completely eradicated if the milk supply were free from tubercle bacilli This can be achieved in two ways either by stamping out the disease from the dairy herds or by killing the tubercle bacilli in infected milk by pasteurisation In order to stamp out in addition to tuberculosis brucella and other infections conveyed by milk the only safe way is by universal pasteurisation Isolation of all open cases would probably

prove an effective means of stamping out the disease caused by human tubercle bacilli but this is not possible Unsuspected cases of open pulmonary tuberculosis in the general wards of a hospital especially in surgical wards are a source of danger to nurses and students All patients admitted to hospital should have a chest examination by miniature radiography as soon after admission as possible No nurse who is Mantoux negative should look after patients suffering from pulmonary tuberculosis Strict precautions should be taken to protect nurses from infection Nurses must wear masks and gowns when carrying out any nursing treatment Patients must wear a mask while the chest is being examined The sputum should be collected in a glass jar or a polythene sputum flask containing 2 fl oz of Miltherex This liquefies the sputum and kills the tubercle bacilli

Prophylactic inoculation of infants with vaccines made from attenuated living bovine bacilli such as the B C G (*bacille bilié* Calmette Guérin) is now being practised extensively B C G is used for vaccination of Mantoux negative medical students and members of hospital staffs and for children born of tuberculous parents The vaccine must be used within 14 days of its preparation It must be shaken well before use and 0 1 ml of the vaccine containing mg 0 5 per ml is injected strictly intradermally using a tuberculin syringe fitted with a Luer needle No 25 or 26 (B W G ) The skin of the forearm is used and a weal about 5 mm in diameter is produced An infant born of a tuberculous mother must be taken away from the mother at birth or if taken away later observed for 6 weeks after separation to make sure it does not develop tuberculosis The vaccine is then given and the infant is kept away from a tuberculous parent for 3 months to allow immunity to develop An adult must be segregated from exposure to tuberculosis for 6 weeks after vaccination The Mantoux test is then repeated and should be positive to 1/100 O T Open cases of tuberculosis in contact with children constitute a grave source of danger

Mass miniature radiography used for selected groups of adults such as factory workers and those in the Services has revealed an incidence of about 4 per 1 000 unsuspected cases of pulmonary tuberculosis In all such cases investigations should be carried out to determine whether or not the disease is active

*Curative* The three cardinal principles of treatment rest fresh air and good food are still of fundamental value There are however no fixed rules for treatment each case presents an individual problem requiring expert knowledge for its solution The patient should be put to bed and kept there until the temperature is normal The usual time in bed is about 3 months He is then gradually allowed up first for toilet purposes then for ½ hour daily and the time up is gradually increased until he is up 5 or 6 hours a day when he may be transferred to a sanatorium to complete his treatment

*Antibacterial drugs* The antibacterial drugs used in the treatment of tuberculosis are streptomycin isoniazid and para aminosalicylic acid None of these should be given alone owing to the risk of the rapid appearance of drug resistant bacilli in the sputum

*Streptomycin* This is injected intramuscularly in the form of streptomycin sulphate In a severe case 1 G should be given daily for 4 to 8 weeks followed by 1 G 2 or 3 times a week In less severe cases it is given 2 or 3 times a week from the onset of the treatment With these doses there is little risk of labyrinthine disturbances or of deafness

*Isoniazid* (Isonicotinic acid hydrazide I N A H ) This is given by mouth as a tablet in doses of mg 100 2 or 3 times daily, according to the weight of the patient combined with streptomycin or with P A S The combined treatment should be continued for at least a year after the last positive sputum culture after the last X ray indicative of improvement and after the last cavity has closed This usually means 18 months to 2 years treatment I N A H may produce peripheral neuritis or rarely a toxic psychosis

*Para aminosalicylic acid* (P A S ) This is given as the sodium salt The patient is not usually able to take more than 12 G in the 24 hours as it is very liable to produce gastro intestinal disturbances It can be administered as enteric coated granules (Bactylan) containing 50% by weight of the sodium salt the content of the measure supplied being *equivalent to 2 G P A S or as enteric coated dragées (Bactylan)* containing 0 69 G of the sodium salt

I N A H and P A S may be given together instead of using streptomycin but it is usual to ring the changes on a combination of two out of the three drugs

If the patient becomes resistant to streptomycin and I N A H *Viomycin* may be injected intramuscularly in doses of 2 G 2 or 3 times a week together with daily doses of P A S Viomycin is often used when preparing a patient for an operation

*Artificial pneumothorax* This is becoming less frequently used but it is of value in some cases combined with antibacterial drugs to close a cavity and so to prevent or retard the appearance of drug resistant bacilli

*Pneumoperitoneum* When effective this produces a reduction of up to 40% of lung volume It is of value in bilateral cases

*Extrapleural compression* Plombage may be effected by the inser*tion of lucite balls or plastic sponges extrapleurally* after the apex of the lung has been freed

*Thoracoplasty* This is of value to close cavities in long standing disease especially in older patients It should be limited to 4 to 7 ribs and done in three stages

*Pulmonary resection* After a full course of antibacterial treatment there may be residual localised solid or cavitated pulmonary lesions especially blocked cavities These may be removed by segmental resection or by lobectomy The patient should remain in bed for 4 to 6 months after the operation to prevent relapse

*Treatment of Certain Complications* Night sweats are at times very distressing *The bed clothes should be light and the windows* kept well open at night A pill containing Zn oxid gr 2 and ext belladon sicc gr ¼ at night should also be tried Hæmoptysis The

treatment is considered on p 189 Laryngitis The patient must neither speak nor whisper and silence should be preserved for periods up to 6 months all communications being made by signs or by writing He must not smoke Streptomycin combined with I N A H is the most efficient treatment An inhalation of a few drops of a solution placed on the pad of a Burney Yeo mask every hour may be used continuously while the patient is awake The following solution is frequently used Ol cassiæ ol eucalypt ol pini creosoti aa partes æquales If there is severe pain on swallowing a powder composed of equal parts of benzocaine and orthocaine may be used As much as will cover a sixpence is placed on the palm of the hand and inhaled directly into the larynx through a curved Leduc s glass tube just before a meal In other cases when the pain prevents swallowing even liquids relief may be obtained by using a glass tube which dips into the milk which is placed on the floor the patient sucking it up with his head hanging over the edge of the bed Firm pressure applied over both ears by the palms of the hands may also enable the patient to swallow in comparative com fort If these measures fail alcohol injection into the superior laryngeal nerve may give temporary relief The dying patient may have his last days made comfortable with the help of the following medicine Cocain hydroclor gr $\frac{1}{8}$ morphin hydrochlor gr $\frac{1}{8}$ gin m 60 honey m 60 aquam dest ad fl oz 1 Fl oz 1 t d s

*Sanatorium Treatment* The sanatorium must be properly equipped with a skilled staff and X ray facilities Major surgery is best conducted in a hospital Many sanatoria are now closing down because the results obtained in hospital or at home with antibacterial treatment are so satisfactory that they are no longer required

*The After treatment* When arrest of the disease has been firmly established the question of return to work and the suitability of the employment has to be decided In general all heavy muscular work should be avoided and outdoor occupations are unsuitable unless the patient is protected from the extremes of climate and from getting wet. Indoor occupation is not harmful provided the environment is healthy and the patient can look after himself adequately at home There remain a certain number of patients who do not do well with treatment These are often sent back to their homes eventually to die They are open cases of tuberculosis and undoubtedly are a great risk to others If all open cases of pulmonary tuberculosis were isolated the hope of stamping out this disease would be increased

## Emphysema

**Definition** Dilatation of the pulmonary alveoli

**Varieties** Certain varieties of emphysema are described which will be considered separately These are Acute emphysema which is vesicular or interstitial Chronic emphysema which is hypertrophic or atrophic

### Acute Vesicular Emphysema

**Definition** Sudden over distention of the pulmonary air vesicles which form blebs or bullæ

**Etiology** Acute vesicular emphysema is associated with severe coughing or muscular strain It may be met with in whooping cough asthma or asphyxia In children it may occur adjacent to a consolidated area of lung in bronchopneumonia and sometimes in women in prolonged labour, or in blast injuries of the chest

**Clinical Findings** The symptoms which might be associated with it are masked by those of the primary condition Its presence can sometimes be detected radiologically by a ring shadow indicating a large bulla or pneumatocele

## Acute Interstitial Emphysema

**Definition** A condition characterised by the presence of air in the interstitial tissues of the lungs the air is derived from ruptured pulmonary alveoli It differs therefore from pulmonary emphysema

**Etiology** The air vesicles may rupture with comparatively moderate muscular exertion such as while playing a game of hockey Other causes include a wound of the chest fractured rib or the severe paroxysms of cough in whooping cough or bronchopneumonia It occurs occasionally in pulmonary tuberculosis apart from any effort

**Pathology** The escaped air tracks along the pulmonary roots and may reach the mediastinum or appear as surgical or subcutaneous emphysema in the neck and chest It may also pass into the pleural cavity or into the retroperitoneal tissues

**Clinical Findings** In cases occurring during exercise the patient feels a tightness or pain in the neck and chest and shortness of breath

*On Examination* The crackling signs of surgical emphysema may be felt on the chest or neck If the air has extended into the anterior mediastinum the superficial cardiac dulness is obliterated the heart sounds are distant, and crunching sounds may be heard over the præcordium as the patient breathes and the heart beats

**Course** The air is usually absorbed spontaneously in a few days

**Treatment** The patient is kept in bed He usually prefers to be propped up A hypodermic injection of morphin sulph gr 1/6 will help to check cough and induce sleep In severe cases relief of tension may be obtained by inserting a needle into the suprasternal tissues or by underwater drainage of a tension pneumothorax

## Chronic Generalised Emphysema

**Definition** A condition of generalised dilatation of the alveoli with an insidious onset

**Etiology** Obstruction to the bronchioles is thought to be a more important cause of emphysema than is a constitutional weakness of the elastic tissues of the lungs The obstruction acts as a ball valve the air gets into the alveoli but its passage out is impeded and so alveolar dilatation occurs with weakness of elastic tissue Other factors which are of importance include 1 Excessive inspiratory efforts In chronic bronchitis and asthma the inspiratory efforts are stronger than the expiratory hence the lungs become over distended 2 Violent expiratory efforts These may be associated with chronic cough or asthma

3 Premature ossification of the costal cartilages The chest wall might become fixed in an inspiratory position with secondary emphysema of the lungs Clinically the chest wall is often unduly rigid in emphysema

Pathology Both lungs are affected They are not hypertrophied but stretched At autopsy they are bulky pale and soft and lacking in elasticity Bullæ may be seen on the surface especially along the anterior borders On section the lungs are pale and the dilated alveoli may be noticeable They float in water As the alveoli dilate the dividing septa break down and bullæ form Aeration of the blood is diminished in emphysema for the following reasons The aerating surface is reduced owing to the loss of the septa The lumen of the vessels is narrowed in the stretched alveolar walls thrombosis occurs in the pulmonary arterioles and so circulation is impeded Some blood is short circuited from the pulmonary arterioles to the bronchial venules The right side of the heart dilates and the motor power of the pulmonary circulation is reduced

Clinical Findings The patient is usually a male of middle age or over He may give a history of winter cough or of asthma His chief complaint is shortness of breath on exertion There may be headache giddiness and loss of appetite

*On Examination* The patient may be cyanosed slightly or to an extreme degree and the fingers may be clubbed The chest Inspection The diameter is increased antero-posteriorly (barrel shaped chest) the ribs run more transversely than normal the subcostal angle is wide and there is kyphosis in the upper dorsal region Post mortem and X ray examinations have shown that marked emphysema may occur without the presence of the barrel shaped syndrome and *vice versâ* Expansion is poor the movement on inspiration being chiefly one of elevation Dilated venules may be seen along the line of the diaphragmatic attachment Palpation Tactile fremitus is diminished The cardiac apex cannot be felt Percussion The note is hyperresonant generally The areas of cardiac and hepatic dulness are reduced Auscultation The breath sounds are weak Expiration may be prolonged scattered rhonchi or râles may be heard There is no bronchophony and no whispering pectoriloquy The heart sounds usually have their maximum intensity over the xiphoid The patient is often unable to blow out a lighted candle If the intrapleural pressure is taken it is often found to be raised X ray examination The translucency of the lungs is increased especially at the bases The ribs run more transversely than normal The diaphragm is low and shows diminished movement The heart shadow is small as the heart rotates to the right with the depression of the diaphragm There is an increased air space in front and behind the heart The sputum This is scanty and rather frothy The blood may show an excess of red cells

*Respiratory function tests* *The vital capacity* This is determined by the use of a spirometer A normal young adult can expire about 3 800 to 4 000 ml after a deep inspiration In emphysema this volume may be reduced by 50% or more Further, a normal person should be

able to expel 95% of his vital capacity air in 3 seconds In emphysema this time is prolonged *The residual lung volume* This is the amount of air remaining in the lungs after a maximal expiration It is increased in emphysema *Spirograms* These are tracings of respiration made on a recording revolving cylinder the writing pen of which is connected with the spirometer whose movements are thus registered In emphysema although the movements in normal respiration are normal in forced expiration after a maximum inspiration there is delayed expulsion of air so that the expiratory curve is broad and shallow

**Differential Diagnosis** The diagnosis of emphysema which is often made with such certainty clinically has been shaken by post mortem observations in which no such condition has been found to exist The X ray findings are most reliable

**Course and Complications** Emphysema tends to be progressive Chronic bronchitis asthma dilatation of the right side of the heart, tricuspid regurgitation a pulsating liver and other signs of heart failure are liable to occur (see Cor pulmonale p 235) Other complications include pulmonary tuberculosis spontaneous pneumothorax or acute interstitial emphysema due to rupture of a bulla

**Prognosis** There is usually no immediate danger, apart from the presence of heart failure The ultimate outlook is very unfavourable The ability to adopt a suitable mode of life very materially improves the prognosis

**Treatment** *Prophylactic* Occupations involving strain should be avoided by those predisposed to or with early signs of emphysema

*Curative* There is no curative treatment All that can be hoped is to decelerate the natural progress of the disease If possible the patient should winter out of Europe in South Africa or Egypt If this is not feasible, every effort should be made to avoid bronchitis and bronchospasm and if they occur they should be treated early and rigorously An emphysema elastic abdominal belt and expiratory breathing exercises as for asthma are of some value Heart failure must be treated by rest venesection digitalis and a low salt diet

Administration of oxygen in severe cases of emphysema may be dangerous and cause death from carbon dioxide poisoning The reason is as follows In emphysema the arterial blood contains an excess of $CO_2$ absorbed from the stagnating air in the alveoli The respiratory centre which is normally stimulated by $CO_2$ eventually becomes insensitive to this stimulus and the respiratory stimulation is now due to hypoxæmia The lack of oxygen in the blood stimulates respiratory movements through the carotid bodies If oxygen is now administered this stimulation to respiration is removed Respiration fails and a great excess of $CO_2$ accumulates in the arterial blood which proves fatal Oxygen should therefore be administered with caution to relieve cyanosis with a flow of 1 to 3 litres a minute using a mask rather than an oxygen tent If symptoms of CO poisoning occur, such as sweating jerky muscular contractions or mental disturbance the oxygen must be discontinued temporarily or the rate of administration reduced

## Localised Emphysema

*(Compensatory or Secondary Emphysema)*

**Definition** Dilatation of the alveoli in localised areas of the lungs

**Etiology** Compensatory emphysema is a secondary process around areas of consolidated fibrosed or collapsed lung It is thus associated with pneumonia or bronchopneumonia new growths an inhaled foreign body fibrosis, bronchitis and pleural effusion It may also develop in the remaining lung after lobectomy or pneumonectomy with consequently increasing dyspnœa A previously healthy lung will usually expand to fill the additional space without the development of emphysema

**Pathology** Inspiratory efforts associated with obstruction to the air entry of a localised area of lung will lead to alveolar dilatation of neighbouring parts of the lung If one lung is largely put out of action as by tuberculous fibrosis compensatory emphysema will occur in the other There is no true hypertrophy of lung tissue but changes similar to those described on p 171 are seen The process therefore diminishes the aerating power of the lung

The condition calls for no special treatment but it is a contra indication to thoracoplasty or pneumonectomy

## Atrophic Emphysema

*(Senile or Postural Emphysema)*

**Definition** Enlargement of the pulmonary alveoli due to degeneration of their septa

**Etiology** Atrophic emphysema is a variety of senile degeneration The alternative view is that the emphysema results from rigidity of the spine and kyphosis enlarging the antero posterior diameter of the chest

**Pathology** The lungs as seen at autopsy, are small dark and friable Small bullæ may be present on the surface and on section the enlarged alveolar spaces are seen The emphysema is non obstructive

**Clinical Findings** The patient who is usually over the age of 60 complains of progressively increasing shortness of breath on exertion and usually he has cough and expectoration due to associated bronchitis

*On Examination* The patient is usually thin and wasted The chest is flat and expansion is poor The tactile fremitus is diminished The percussion note is hyperresonant but the cardiac and hepatic dulness are not diminished to any degree The breath sounds are weak and expiration is a little prolonged Adventitious sounds due to bronchitis may be heard The diaphragm may be higher than normal

**Treatment** No special treatment is available beyond the care necessary for an elderly patient who is liable to bronchitis or to dilatation of the heart

## Tumours of the Lungs

**Simple Tumours** Those which arise in the bronchi have been considered on p 146 *The hamartoma* is a common benign tumour It

is composed of cells derived from the various embryonic layers and only contains structures normally found in the organ in which it arises The cartilage in it may be impregnated with calcium giving a dense shadow on X ray examination Other simple tumours include a fibroma, lipoma, chondroma, leiomyoma and hæmangioma Arterio venous fistulæ may also occur in the lungs giving rise to cyanosis and clubbing of the fingers A murmur is heard over the affected portion of lung and an X ray film shows a round or lobulated shadow in the lung field

**Malignant Tumours** *Primary carcinoma of the lungs* arises in a bronchus and is described on p 147 *Secondary carcinoma* The primary growth may be in the breast alimentary tract, kidney prostate and less frequently in other sites It is usually bilateral, giving rise to somewhat ill defined opacities in the lung fields as seen on X ray examination The patient may complain of shortness of breath, loss of weight and malaise In some cases a bilateral pleural effusion forms Sarcoma or endothelioma may be primary or secondary, whereas hypernephroma malignant deciduoma and seminoma are secondary tumours *Primary sarcoma* This is a rare, solitary and rapidly growing tumour occurring in early life *Secondary sarcoma* The primary growth is in bone or in a melanotic tumour *Secondary lymphosarcoma* In addition to the growth in the lungs enlarged glands are usually found elsewhere as in the neck and axillæ and diagnosis is made by gland section *Secondary teratoma and seminoma* The primary growth is in the testicle the secondary growths in the lungs are described as cannon ball " tumours on X ray examination

## Fibrosis of the Lungs

**Definition** An excess of fibrous tissue in the lungs

**Etiology** The fibrosis is usually productive in origin being formed in response to irritation , less frequently areas of replacement fibrosis occur

**Pathology** The fibrous tissue may be distributed in various ways in the lungs It may be diffuse as in tuberculosis sarcoidosis pneumo koniosis and in chronic passive hyperæmia Generalised fibrosis affecting especially the lower lobes may be met with in generalised scleroderma This may lead to cystic changes Lobar fibrosis may follow lobar or bronchopneumonia Peribronchial fibrosis is met with in chronic bronchitis Localised fibrosis occurs around a tumour cyst, abscess or granuloma of the lungs, and after bronchopneumonia Pleurogenous fibrosis is secondary to chronic pleurisy fibrous tissue extending into the subjacent lung When the fibrosis is localised a depressed thickened area may be seen and felt This is often found at the apex of a lung due to a healed tuberculous focus With diffuse fibrosis the lung is contracted firm and darker than normal There are often pleural adhesions On section the lung is tough the strands of fibrous tissue are apparent and there may be bronchial dilatation

**Clinical Findings** It is only in the diffuse variety that symptoms are likely to be noted The patient complains usually of shortness of breath on exertion with cough and expectoration especially in the winter

Occasionally unilateral fibrosis will cause dysphagia by torsion on the œsophagus

*On Examination* In a long standing case there is usually some *cyanosis of the face and clubbing of the fingers* If the fibrosis is unilateral there will be flattening and diminished expansion on one side of the chest and the cardiac impulse may be seen displaced towards the affected side The corresponding shoulder may be low and some scoliosis present If the fibrosis affects the left upper lobe cardiac pulsation is usually seen in the second and third left intercostal spaces close to the sternum due to uncovering of the heart Palpation The diminished expansion is confirmed and tactile fremitus is lessened Percussion The note is impaired over the affected lung and it may be hyperresonant on the other side Auscultation The breath sounds are weak over the fibrosed lung and harsh on the opposite side Some leathery fibroid rales may be heard and frequently rhonchi are present in both lungs Vocal resonance is diminished and there is no whispering pectoriloquy If areas of consolidation or excavation are present the *breath sounds are more bronchial in type and voice conduction is* increased Basal fibrosis is often seen in children after broncho pneumonia The heart is slightly displaced to the affected side the note at one base is impaired the air entry weak and fine to medium rales are heard constantly on deep breathing or after cough

**Differential Diagnosis** The diagnosis of pulmonary fibrosis does not usually present difficulties but it may be impossible to be certain as to its causation A Wassermann test should be done and if positive the effect of a course of antisyphilitic treatment determined The sputum should be examined several times for tubercle bacilli and the nature of the predominating organisms investigated The occupational history of the patient and the X ray findings may point definitely to pneumokoniosis Fibrosis of a lower lobe is often the result of pneumonia bronchopneumonia or pleurisy This is *especially liable to occur in children after bronchopneumonia associated* with measles or whooping cough

*Diffuse interstitial pulmonary fibrosis* or the *Hamman Rich syndrome* is a disease of unknown etiology It has been suggested that it is a localised form of collagen disease It is characterised by progressive dyspnœa cyanosis cough pain in the chest and eventually right sided heart failure Diagnosis may be made in life by lung biopsy the microscopical changes being characteristic Fibrous tissue forms in the alveolar walls and later spreads to invade the lungs The condition is progressive and death usually occurs within 12 months of the onset of symptoms

**Course and Complications** Fibrosis is usually slowly progressive It may lead to bronchiectasis and be accompanied by areas of com pensatory emphysema

**Prognosis** In tuberculosis fibrosis is welcomed as *an indication* of arrest of the disease Apart from this fibrosis limits the functions of the lungs and increases the strain on the heart and so tends to shorten life It may also conduce to bronchiectasis

Treatment *Prophylactic* Adequate inspiratory breathing exercises should be carried out during convalescence in every case of bronchopneumonia, pneumonia and empyema to expand the base of the lung and prevent fibrosis

*Curative* In basal fibrosis non tuberculous in origin inspiratory breathing exercises should be performed daily to expand the lung and endeavour to prevent the development of bronchiectasis Dusty environments should be avoided The patient must live well within the limits of his respiratory and cardiac reserves and climatic treatment in the winter is of value in preventing bronchitis

### Pneumokoniosis

*(Dust Disease of the Lungs)*

Definition Fibrosis of the lungs due to inhalation of dust

Etiology The following varieties are described according to the nature of the irritant *Anthracosis* (coal) *Silicosis* (silica as in the following occupations —Gold tin zinc or hæmatite iron ore and coal mining sandblasting flint and pebble crushing the manufacture of abrasive soaps metal grinding slate quarrying granite sandstone and pottery work) Workers in mica talc and those exposed to bauxite fumes *Lithosis* or *chalicosis* (stone particles) *Siderosis* (tin copper lead or iron) this is possibly silico siderosis *Asbestosis* (metallic particles containing silica and iron and also vegetable fibres) Asbestos workers are employed in making matches filters paints roofing tiles brake linings etc *Byssinosis* (cotton particles) *Bagassosis* The bagasse dust which contains about 6% of silica may be inhaled in *bread making factories Bagasse is sugar cane* which has been broken down and from which sugar has been extracted *Berylliosis* Beryllium is used in the manufacture of fluorescent lamps In chronic beryllium poisoning nodules appear in the lungs Berylliosis behaves like a granuloma and dissemination occurs in the liver *Farmers lung* This *is due to inhalation of dust from hay and grain*

Pathology When particles of dust are inhaled for long periods fibrotic and bronchitic changes ensue in the lungs It is very doubtful whether in man the particles reach the lungs from the alimentary tract and the lymphatic system Coal dust particles are to a certain extent *expectorated but silica particles tend to remain in* the lungs and pulmonary tuberculosis is very prone to supervene Tuberculosis may occur in coal miners but it is common in gold miners and it also occurs in some cases of asbestosis Dusts containing free crystalline silica (SiO ) are dangerous providing the particles are smaller than 10 microns in diameter and their inhalation leads to the formation of silicotic nodules and to fibrosis Tuberculosis may supervene It is now believed that the silica produces fibrosis not by mechanical irritation but rather as the result of the solubility of the silica The lesions are nodular and distributed throughout the lungs It has been shown experimentally that the evil effect of quartz dust is reduced by coal dust and practically abolished by shale This is probably due to

the fact that the solubility of the quartz is reduced by coal because aluminium is released which forms a covering over the quartz particles and so lowers their solubility Silicosis is not uncommonly combined with anthracosis in coal miners lungs Anthracosis occurs in two forms In the simple variety there are minute nodules chiefly in the upper and middle zones of the lungs In the complicated form there are large localised opacities seen by X rays and tuberculous fibrosis develops The outlook in this variety is very unfavourable In anthracosis the lungs as seen at autopsy are black in silicosis they are grey and in siderosis they are brown They are firm and contracted due to nodular or diffuse fibrosis and the pleura is thickened with adhesions The bronchi are often dilated and the root glands are enlarged hard and pigmented In asbestosis the silica is combined with such bases as magnesium iron calcium sodium and aluminium and there is a diffuse interstitial fibrosis of the lower lobes Examination of the lungs of silver finishers who inhale iron oxide dust has shown that the stippled or reticular X ray shadows are due to the aggregations of iron oxide dust and not to fibrotic changes

**Clinical Findings** The patient is usually a male between the ages of 20 and 40 who gives a history of having worked for some years (usually over 6) in one of the dusty occupations mentioned above He complains of cough and expectoration with shortness of breath and later of lassitude and loss of weight

*On Examination* In the early stages the only abnormalities are those detected by the X rays In silicosis small nodules of fibrous tissue may be seen in the lung fields with diffuse streakiness of the lungs and heavy root shadows The nodular shadows become more obvious as the condition progresses and later a diffuse mottling occurs chiefly in the central zones of the lungs In asbestosis there is a reticular fibrosis The physical signs in an established case are chiefly those of chronic bronchitis and emphysema the air entry over both lungs being weak The sputum in asbestosis may contain micro scopical golden yellow asbestosis bodies with bulbous extremities consisting of a central core of asbestos surrounded by discs which give the Prussian blue reaction for iron and are probably composed of iron silicate

Some cases of acute silicosis have occurred in workers in a factory manufacturing a cleaning powder containing ground silica sodium carbonate and soap The alkali is probably responsible for the rapid onset of the symptoms Death occurred in two cases and in one of them tuberculous lesions were also demonstrated in the lungs Cases have also been described in tunnellers and in rock drillers in lead mines The symptoms of farmers lung resemble those of asthma

**Differential Diagnosis** The diagnosis of silicosis can usually only be established with certainty by X ray examination of the lungs

**Course and Complications** The course is progressive unless arrested by removal of the patient from the noxious environment Complications include bronchitis bronchiectasis emphysema tuberculosis and cardiac dilatation Silicosis is occasionally followed by carcinoma of a bronchus

There is not sufficient evidence to say whether asbestosis predisposes to tuberculosis, although it undoubtedly occurs in certain cases

**Prognosis** This is unfavourable unless the patient is removed from his dusty occupation in the early stages of the disease

**Treatment** *Prophylactic* Measures should be taken in all dusty occupations to prevent inhalation of the dust These include the use of respirators water sprays incorporated in the coal drills, and fans The workers should be examined and X rayed every 6 months

*Curative* The patient should be removed from his occupation The treatment then is as for chronic bronchitis and emphysema Berylliosis may be treated with cortisone or ACTH

### Syphilis of the Lungs

The condition is usually congenital, rarely acquired

**Pathology** In congenital syphilis a condition of "white pneumonia is usually present A lobe or the greater part of one or both lungs may be involved The affected portion is enlarged and depressions caused by the pressure of the ribs may be seen on the surface The lung appears pale and dry and sinks in water On section the lung may somewhat resemble the tissue of the pancreas ( pancreatisation ") The alveolar epithelium is degenerated and the alveoli contain some leucocytes There is round celled infiltration of the interalveolar tissue with numerous small gummata The Treponema pallidum is present In acquired syphilis the lesion may take the form of a gumma

**Clinical Findings** The infant with congenital syphilis of the lungs is still born, and the lesion is demonstrated at autopsy or death occurs after a few days during which signs of pulmonary consolidation may have been detected The Wassermann reaction of the blood both of the mother and child, is generally positive and other stigmata of congenital syphilis are noted In adults the clinical findings are those of a local pulmonary tumour or of a blocked bronchus if the gumma is situated intrabronchially Bronchoscopy the positive Wassermann reaction and the response to anti syphilitic treatment are of value in diagnosis

### Actinomycosis of the Lungs

**Etiology** The causative organism is the *Actinomyces bovis (Strepto thrix actinomyces or ray fungus)* This may gain access to the lungs from the teeth or tonsils

**Pathology** Granulomatous lesions are found in the lungs often in the lower lobes Suppuration and fibrosis occur around them and they may spread to involve the pleura chest wall or liver

**Clinical Findings** The history of the case may closely resemble that of chronic pulmonary tuberculosis or of a neoplasm of the lungs Thus the patient complains of cough expectoration which may be bloodstained progressive loss of weight and irregular fever The first symptom may be pleural pain

*On Examination* The physical signs in the lungs are very variable Thus they may be those characteristic of bronchitis or of an area of

consolidation or excavation of the lung In other cases there is dry or wet pleurisy The lesion may ulcerate through the chest wall producing a purulent discharge containing ' sulphur granules The sputum should be specially examined for the ray fungus by the appropriate staining methods

**Differential Diagnosis** This may be very difficult before the organism is found in the sputum or pleural fluid The X ray findings often closely resemble those of chronic pulmonary tuberculosis or of carcinoma of the lung

**Course and Complications** The course is usually slowly progressive the infection may spread elsewhere especially to the liver or chest wall

**Prognosis** The outlook has been improved by modern treatment

**Treatment** A prolonged course of treatment with penicillin should be given injecting intramuscularly one million units every 6 hours for 1 to 2 months Sulphadiazine may be combined with the penicillin treatment 1 G t d s for 14 days If this fails potassium iodide should be given in increasing doses beginning with gr 5 t d s and working up to gr 60 or 90 t d s Cutaneous sinuses may be treated with deep X rays

## Aspergillosis of the Lungs

**Etiology** Infection with a fungus the Aspergillus fumigatus The infection is spread by grain

**Clinical Findings** The patient is usually a miller farm worker or pigeon breeder He complains of symptoms resembling those of chronic pulmonary tuberculosis more rarely he is acutely ill

The diagnosis can only be made if the organism is found in the sputum The acute case is liable to be mistaken for lobar pneumonia In a case recorded in 1945 the sputum contained whitish flecks composed of masses of mycelial threads and no other organisms Rapid recovery followed the administration of potassium iodide sulphonamides having failed to effect any improvement

**Treatment** This consists in administering potassium iodide as for actinomycosis

## Moniliasis (Candidiasis) of the Lungs

**Etiology** The causative organisms are various fungi of the monilia group It is met with among tea tasters in Ceylon Monilia are often present in the bronchi of apparently healthy individuals or in chronic lung disease such as bronchitis They may become pathogenic when other bacterial flora have been eliminated by the use of antibiotic drugs

**Clinical Findings** The patient complains of symptoms resembling those of chronic bronchitis and the causative organisms are found in the sputum

**Treatment.** This consists in the administration of potassium iodide in gradually increasing doses from gr 5 to gr 60 t d s

## North American Blastomycosis

**Etiology** *This is a systemic infection with the Blastomyces dermatitidis* which occurs only in the yeast form in human beings It is widespread in the United States and in Canada

**Clinical Findings** In the pulmonary variety the patient has a cough and sputum X ray examination shows shadows suggesting confluent *pneumonic areas often with cavitation The skin is frequently* involved chronic abscesses forming on the face, hands wrists and ankles The lesions may be widely disseminated throughout the body The diagnosis is made by finding the causative organism in the sputum or skin lesions The disease is usually fatal within a few years

**Treatment** *Stilbamidine may be given by slow intravenous* injection After a preliminary test of mg 50 mg 150 are injected daily in 300 ml 5% dextrose solution The course usually lasts about a month In some cases this is followed by trigeminal neuralgia

## Collapse of the Lungs

### (*Pulmonary Atelectasis*)

**Varieties** *Congenital pulmonary collapse* is met with in still born infants or in those in whom the respiratory efforts are very feeble or where there is obstruction to the air way

*Acquired pulmonary collapse* is of two varieties relaxation and compression collapse and absorption collapse

## Relaxation and Compression Collapse

***Etiology*** *Relaxation collapse may be due to conditions which* reduce the size of the thoracic cavity such as a thoracoplasty diaphragmatic hernia diaphragmatic paralysis following a phrenic crush a pleural or pericardial effusion or a pneumothorax in which the intrapleural pressure is below zero In these cases the elastic recoil of *the lungs comes into increased action Compression collapse* results from a pleural effusion or pneumothorax in which the pleural pressure is positive

## Absorption Collapse

**Etiology** This results from bronchial obstruction the air in the alveoli beyond the blockage being absorbed and *the portion of lung* supplied by the bronchus collapsing The obstruction may be within or without the bronchus A foreign body such as an acorn may lodge in the main bronchus the whole lung collapsing The obstruction is more often caused by plugs of mucus in which case a segment a lobe or *the whole lung may be affected* This may occur in acute bronchitis in influenza or after an operation especially on the upper abdomen It is also met with in post diphtheritic paralysis in poliomyelitis after injection of Lipiodol into healthy bronchi and in association with hæmoptysis Other causes include a new growth or gumma of a bronchus the pressure of an aneurysm fibrosis or consolidation of the lung When fluid replaces air in the partially collapsed segment or lobe

a *consolidation collapse* results and this may lead to an *infected atelectasis*

**Clinical Findings** The signs of local areas of collapse are dulness and weak air entry These are often met with in influenza and change rapidly from day to day as the collapsed portion of lung re expands and another zone collapses

An important clinical condition which may occur in children or young adults is due to infected mucus from the naso pharynx being inhaled into a bronchus The patient is suddenly taken ill with a high fever 103 or 104° F and careful clinical examination will reveal a small area of weak breath sounds with perhaps a few rales

Segmental areas of collapse give rise to small linear shadows on X ray examination Collapse of the right middle lobe is characterised by a triangular shadow seen best in the lordotic and lateral X ray films

**Treatment** Re expansion of the collapsed area of lung rapidly follows expectoration of the obstructing mucus plug Tilting over a chair deep breathing coughing slapping the chest over the affected side rolling the patient from side to side and the inhalation of 5% carbon dioxide and 95% oxygen from a cylinder through a gas bag and face mask are all measures which may be employed Bronchoscopic aspiration is rarely necessary

## Massive Collapse

**Etiology** The whole lung or a lobe may collapse It commonly follows operations especially upon the upper abdomen and is known as *post operative massive collapse* It also occurs after injuries to the abdomen or legs in which the chest is not involved It may be met with in one lung when there is a non penetrating wound on the opposite side of the chest

The reason why it occurs after major upper abdominal operations is because the patient is prevented from breathing deeply with the lower lobes and from coughing up mucus by tight abdominal bandages by pain resulting from the operation and by the calming effect of sedative drugs This allows mucus to accumulate in and obstruct a main lobar bronchus Some cases may be due to nervous reflexes inhibiting the movements of the diaphragm or intercostal muscles or resulting in reflex constriction of the bronchioles with inspiration expiration being normal so that the lung is deflated with respiration

**Clinical Findings** The patient is cyanosed in some instances In the early stages the affected side of the chest is immobile and the air entry is very weak over the affected lung usually over the lower lobe The heart is displaced to the affected side The temperature and pulse and respiration rates are raised Later there is dulness over the affected lobe with bronchial or tubular breathing whispering pectoriloquy and bronchophony and a few fine rales The breath sounds over the opposite lung are harsh and the percussion note may be hyperresonant There is usually some frothy expectoration X ray examination shows displacement of the mediastinum to the affected side with elevation of

the diaphragm The ribs become approximated, giving a tiled appearance The collapsed lung casts a shadow which may be sufficiently dense to obscure the ribs

**Differential Diagnosis** Before massive collapse was clearly described by Pasteur at the *Middlesex Hospital London in 1908 it* was usually mistaken for post operative pneumonia The chief distinguishing features are the homolateral cardiac displacement the absence of rusty sputum and labial herpes, and the course of the illness, which is more rapid than that of pneumonia The diagnosis is confirmed by X ray examination by which the collapsed part of the lung and the raised cupola of the diaphragm are revealed The chief conditions which have to be excluded are *pulmonary thrombosis or* embolus and in left sided affections, a spontaneous pneumothorax as the stomach resonance with a massive collapse may extend up as high as the fourth rib This gives a hyperresonant note which may be thought to be due to air in the pleura

**Course and Complications** In about 2 or 3 days the affected lung may re expand the dyspnœa cease and the temperature and pulse and respiration rates fall to normal Pleurisy bronchitis pneumonia, bronchiectasis and abscess may occur as complications

**Prognosis** This is usually good unless the collapse is bilateral or due to poliomyelitis or unless pneumonia supervenes

**Treatment** *Prophylactic* In any case in which there are signs of bronchitis the patient should be given a course of penicillin injections for a few days before the operation He should be encouraged to take periodically a few deep breaths after the operation The inhalation of 5% carbon dioxide in 95% oxygen for a few minutes at the end of an anæsthetic assists in ventilating the lungs Changes of position in bed and the avoidance of binders which constrict the lower ribs are of first importance

*Curative* Bandages restricting the lower part of the chest should be loosened and the patient laid on his back flat in the bed and rolled from side to side 12 times This simple manœuvre is often surprisingly successful even when a lobe has been collapsed for several days The *breathing and pulse rapidly subside in rate and the obstructing* plug of mucus may be expectorated Subsequently the patient should inhale a mixture of oxygen and 5% carbon dioxide for several hours Coramine 1 5 ml should also be given subcutaneously every 6 hours if the patient is collapsed If these measures fail a bronchoscopic aspiration may be performed to remove any mucus present Sulphadimidine or penicillin *should be given for a week or so after re expansion has taken place*

## Hyperæmia of the Lungs

There are two varieties of hyperæmia of the lungs or as it is often called of congestion of the lungs active and passive

*Active hyperæmia* This is associated with inflammatory lesions of the lungs, a pulmonary infarct or the inhalation of mustard gas

*Passive hyperæmia* The outflow from the lungs through the pulmonary veins may be diminished in heart failure chronic bronchitis

and emphysema It is met with especially in elderly bed ridden patients constituting hypostatic congestion Infection may supervene with rise of temperature thus resulting in what had been called hypo static pneumonia The pulmonary veins are at times obstructed by enlarged mediastinal glands or by thrombosis

### Hæmorrhagic Concussion of the Lungs

#### (*Blast Injury of the Lungs*)

**Etiology** Hæmorrhagic lesions are often found in the lungs especially in children from peace time injuries such as road accidents in which no injury to the chest wall and no fracture of the ribs can be found Blast from a high explosive bomb may kill without signs of external injury

**Pathology** Hæmorrhagic areas are found usually deep in the lungs and often at the costo-phrenic sinus when the spleen or liver is also damaged Blood stained mucus may be present in the upper respiratory passages There may also be hæmothorax and effusion of blood into the mediastinum

**Clinical Findings** The patient may be found dead as described above, or complain of faintness shortness of breath and pains in the chest or abdomen

*On Examination* Patients are seen to be suffering from varying degrees of shock there is pallor cyanosis a rapid and feeble pulse and low blood pressure In addition there is dyspnœa chiefly of an expiratory type the chest being over distended and bulging The physical signs in the lungs vary the breath sounds are often weak throughout both lungs and a sudden rise of temperature may herald the development of a lobar or lobular consolidation Abdominal pain tenderness and muscular rigidity are met with in some cases suggesting an acute abdominal condition Hæmoptysis of varying degrees may occur and restlessness is a predominant feature in all cases of blast injuries to the lungs Ruptured ear drums are to be expected if the person has been sufficiently close to the explosion to suffer internal injuries from blast

**Treatment** Shock should be combated by placing the patient in a bed heated with hot bottles or an electric cradle Morphin sulph gr ½ should be given subcutaneously to control restlessness and severe pain and if necessary repeated up to a maximum of gr 1 in 24 hours Oxygen should be inhaled from a B L B mask Plasma transfusions of 1 to 2 pints are often helpful but whole blood transfusions are only required if there is anæmia

### Acute Œdema of the Lungs

**Definition** A condition in which the pulmonary alveoli are suddenly flooded with serous exudate

**Etiology** The cause is not known Probably there are several varieties There are three main views 1 The cardiac theory The œdema is considered to be a manifestation of sudden left sided heart failure perhaps due to a disproportion between the effective power of

the ventricles, the left expelling less blood than the right It may occur in association with paroxysmal auricular flutter 2 The toxic theory This may account for certain cases met with in connection with chronic nephritis, pregnancy and infectious diseases in which no cardio vascular lesion is demonstrable 3 The angio neurotic theory This helps to explain the sudden onset of the condition in young people, apparently in perfect health, and is supported by the simultaneous appearance of œdema of the face

Clinically, acute œdema of the lungs is most often associated with arteriosclerosis, aortic disease, myocardial degeneration coronary occlusion pulmonary infarction and chronic nephritis Less frequently it occurs as a complication of aspiration of a pleural effusion

**Clinical Findings** The patient is usually over the age of 40 Apparently in normal health he is suddenly seized with faintness and apprehension and then becomes very short of breath and may rapidly pass into a stage of semi or complete unconsciousness

*On Examination* If the patient is conscious he is found sitting up and alarmed with great distress of breathing The skin is pale and moist, and slight cyanosis is present The heart is usually regular but rapid Rales are heard all over both lungs After a period varying *from a few minutes to half an hour frothy fluid wells up into the mouth* from the lungs, and may stream out through the mouth or nostrils The fluid is often stained pink Transient albuminuria may occur

**Differential Diagnosis** In practice there is little difficulty in *diagnosis the physical signs in the lungs and the frothy expectoration* are characteristic

**Course and Complications** The attack may last for a few minutes or for several hours As many as 70 attacks have been recorded in the same patient

**Prognosis** The first attack may prove fatal the prognosis is always extremely grave if the patient loses consciousness

**Treatment** A hypodermic injection of morphin sulph gr $\frac{1}{4}$ and atropin sulph gr 1/50 should be given immediately The morphine should be repeated in 2 hours if necessary and oxygen administered by nasal catheter or mask If this treatment fails 10 to 15 oz of blood should be removed from a vein in the arm Lumbar puncture is usually of no avail The congestive heart failure should be treated with digitalis by mouth aminophylline 0 25 G in 100 ml sterile water, by intravenous injection by a salt poor diet and diuretics

## Chronic Œdema of the Lungs

*This occurs in association with passive hyperæmia (see p 182)* and as a manifestation of œdema in renal disease

## Infarction of the Lungs

**Definition** Obstruction of a branch of the pulmonary artery with resultant hæmorrhage into the lung alveoli and interstitial tissues

**Etiology** There are two varieties the embolic and the thrombotic

*Pulmonary embolus* This is the most common cause The embolus

may consist of blood clot of a fragment of an intracardiac vegetation less frequently of air of particles of fat new growth of parasites such as a hydatid daughter cyst or of droplets of amniotic fluid containing meconium *Predisposing causes* Cardiac disease such as mitral stenosis, infective endocarditis venous stasis and childbirth Surgical cases are most commonly secondary to operations in which the anterior abdominal wall has been incised This tends to lead to stagnation in the iliac veins and their tributaries owing to inhibition of diaphragmatic respiration caused by pain in the wound with consequent venous thrombosis Pulmonary embolus is also comparatively common in cases of fractured femur probably due to venous stagnation caused by the immobilisation of the limb Fat embolus may also result from a severe shaking multiple and compound fractures severe burns etc Sepsis does not appear to play an important part in the etiology of pulmonary embolus Air embolism has been recorded as a complication of pneumoperitoneum of vaginal douching with soapy water during pregnancy in attempts at abortion air in the form of froth being introduced into the uterus under considerable pressure and of insufflation of the vagina with a silver picrate powder in the treatment of a vaginal discharge It has also occurred during blood transfusion as the result of raising the pressure in the bottle by a Higginson syringe while the filter was partly blocked

*Pulmonary thrombosis* This may occur in any acute or chronic lung disease in mitral stenosis and also as a secondary process in connection with a pulmonary embolus It may also be a manifestation of phlebitis migrans or occur after operations

Pathology The pulmonary embolus when formed of blood clot may be derived from one of the veins in the abdomen or legs It is detached and carried through the right side of the heart to the lungs It may also be formed from blood clot in the right auricle in cases of congestive heart failure or in mitral stenosis Air embolus may result from an operation on the chest wall as in exploring an old empyema track Recent infarcts resemble blood clot whereas old ones become absorbed and replaced by fibrous tissue When the infarct extends to the lung surface it is cone shaped with the base outwards and evidence of dry pleurisy is seen on the surface Deep seated infarcts are oval or circular The infarct sinks in water It is not always possible at autopsy to discover the source of the embolus

Clinical Findings In a typical post operative case there is a history of an operation about 10 days previously The patient is suddenly seized with agonising pain in the side of the chest which is intensified with each inspiration There may also be cough and the immediate expectoration of blood or of blood and frothy sputum The pain may be so severe that the patient collapses

*On Examination* The patient is found sitting up in bed often leaning over towards the affected side much distressed with rapid and shallow respirations There may be no physical signs in the lungs in the early stages beyond generalised weak air entry In a few hours an area may be detected in which there is slight dulness with definitely

deficient breath sounds and a few fine rales as œdema occurs around the infarct Later a pleural friction rub may appear with signs of consolidation of a portion of the lung usually in the lower lobe If the infarct is basal, involving the diaphragmatic pleura, the pain is referred to the tip of the corresponding shoulder The temperature, and pulse and respiration rates are raised All cases do not conform to this type There is the cardiac variety, in which the patient after an operation suddenly becomes pale and collapsed suffers no pain is dyspnœic, and dies in a few minutes Such cases are mistaken for heart failure until the post mortem examination reveals the presence of a pulmonary embolus There is also a cerebal or apoplectiform variety Here the patient who has been convalescing from an operation for about 2 weeks and who perhaps is just getting out of bed suddenly falls unconscious, is cyanosed and has stertorous breathing Death occurs in a few hours and at the autopsy no cerebral hæmorrhage is found, but a pulmonary embolus is present With fat embolism there is a symptom free interval of a few hours to 2 days This is followed by dyspnœa pallor cyanosis restlessness and sweating The patient is very ill and brings up frothy sputum which may be blood tinged Fat globules may be found in the sputum Maternal pulmonary embolism due to amniotic fluid is said to be the commonest cause of death during labour and the following nine hours It is associated with excessive uterine contractions and amniotic fluid containing meconium Lesser degrees of severity may cause post partum shock and collapse

**Differential Diagnosis** This is usually easy when there is severe pain in the chest Hæmoptysis occurring in mitral stenosis is generally considered to be due to an infarct in the lungs rather than to passive hyperæmia

**Course and Complications** The signs of an infarct in the lungs usually persist for about 1 to 2 weeks and the sputum may contain blood for about 7 to 10 days Further infarcts may occur at short intervals If the embolus is infected abscess of the lung may ensue

**Prognosis** This is very grave if the infarct is large and in the cardiac and cerebral types of cases

**Treatment** *Prophylactic* After abdominal operations and in fractures especially of the femur costal and diaphragmatic respiration should be as free as possible and general muscular contractions and leg movements encouraged If venous thrombosis occurs in a limb anticoagulants should be given It is doubtful whether getting the patient up on the second or third day after a major operation has diminished the incidence of post operative thrombosis

*Curative* The immediate treatment of a pulmonary infarct is directed to the relief of pain and the alleviation of shock Pain is most effectually abolished by strapping the affected side of the chest from below upwards in a position of full expiration The strapping should include portions applied across the top of the shoulder from before backwards A hypodermic injection should be given of morphin sulph gr 1/4 and atropin sulph gr 1/120

Anticoagulants should be administered to diminish the coagulability

of the blood *Heparin* may be given intravenously or intramuscularly It is put up in 5 ml rubber capped vials containing 1 000 5 000 and 25 000 international units per ml One hundred units equals 1 mg of heparin

*Heparin* should be injected intravenously directly the patient is seen the dose being 15 000 units Four hours later 10 000 to 12 500 units should be injected intramuscularly with 0 5 ml of 2% procaine mixed with each injection to reduce pain every 8 to 12 hours for 48 hours The clotting time should be prolonged by this method up to about 20 minutes (normal 4 to 7 minutes by the Lee and White method)

*Dindevan* should be given by mouth simultaneously the first dose at the same time as the intravenous injection of heparin and repeated in 12 hours The dose is mg 100 (2 tablets) On the next day mg 50 is given every 12 hours and subsequently mg 25 to 75 daily The amount of Dindevan required is determined by daily estimations of the prothrombin time This should be prolonged to between 30 and 40 seconds From this the prothrombin concentration can be worked out This should be kept at between 20% and 30% If it rises more Dindevan is required if it falls below 20% the Dindevan should be omitted for a day The prothrombin index is the control prothrombin time divided by the patient s prothrombin time multiplied by 100 This should be kept at between 40% and 50% The urine of the patient taking Dindevan may be orange red in colour This is of no significance It should be tested daily for blood

If hæmorrhage occurs the anticoagulant should be stopped If the bleeding is due to heparin the clotting time can be quickly restored to normal by the intravenous injection of 10 ml of 1% protamine sulphate solution Hæmorrhage due to Dindevan should be treated by the intravenous injection of mg 10 of vitamin K (Synkavit) or mg 20 to 25 can be given by mouth Alternatively a blood transfusion of 1 pint may be given

In cases in which the heart has stopped beating immediate embolectomy has been performed with some success The base of the pulmonary artery is opened and the clot removed

## Hæmoptysis

**Definition.** Spitting of blood

**Etiology** Hæmoptysis may be true or spurious In true hæmoptysis the blood is derived from the larynx trachea bronchi or lungs whereas in spurious hæmoptysis the source of the bleeding is above the larynx

The causes are very numerous *True hæmoptysis* Pulmonary tuberculosis is the most frequent cause next to which come mitral stenosis associated with pulmonary infarct and congestive (left ventricular) failure Other fairly common causes are pneumonia bronchopneumonia infarction from any cause and bronchiectasis Less frequently hæmoptysis is due to a tumour of the bronchus such as an adenoma carcinoma or angioma secondary carcinoma

hereditary telangiectasis abscess or gangrene of the lungs bronchitis spirochætal bronchitis paragonomiasis actinomycosis hydatid infection bilharziasis, pneumokoniosis or erosion of the lung by a pneumolith (*hæmoptysis calculosa*) Penetrating wounds of the lungs due to a foreign body or fractured rib or due to puncture of the lung in pneumothorax treatment are sometimes met with Hæmoptysis may also be due to blood diseases such as purpura, leukæmia hæmophilia, or pernicious anæmia to deficiency diseases such as scurvy or lesser degrees of vitamin C insufficiency to lack of vitamins P or K or to the hæmorrhagic forms of small pox and measles It is at times associated with a high blood pressure, arteriosclerosis polyarteritis nodosa and emphysema There is probably no such thing as hæmoptysis due to vicarious menstruation An aneurysm may erode into a main bronchus or into the trachea and cause a recurrent weeping ' of blood or a fatal hæmorrhage Other tracheal and bronchial causes include tumours, the infective granulomata such as tuberculosis or syphilis and erosion of a caseous tuberculous gland

Hæmoptysis in children or young adults is occasionally due to *idiopathic pulmonary siderosis* The clinical picture is that of a child with anæmia the cause of which is obscure The sputum does not usually contain fresh blood but is reddish brown due to hæmosiderin It is thought that the disease may be due to an increased permeability of the pulmonary capillaries There is damage to the elastic tissue of the lungs with deposits of hæmosiderin and fibrotic changes in the lungs

*Spurious hæmoptysis* The bleeding comes from the gums, from pharyngeal varices or from the nose It is sometimes self induced and a form of malingering Betel nut chewing in India and Ceylon may simulate hæmoptysis

Clinical Findings *These vary with the amount of* blood expectorated Thus the sputum may be stained with blood small dark clots may be expectorated or there may be a frank hæmoptysis of bright frothy blood of several ounces, or a profuse discharge of a pint or more of blood may occur The sputum usually contains blood for 2 or 3 days the colour becoming darker after the bleeding has stopped and stale blood is expectorated Unless the bleeding is severe the patient is unaware of it until he sees the sputum In a sudden hæmoptysis, in which pure blood comes up he may just ' clear his throat " taste and feel a saltish warm sensation in the mouth and then spit out the blood Rupture of a vessel into a pulmonary cavity may cause death in a few minutes whereas if an aneurysm bursts into a bronchus the patient falls dead in a few seconds In pulmonary tuberculosis hæmoptysis is more common in hot weather and in the majority of cases it occurs when the patient is at rest In some cases hæmoptysis leads to lobar collapse of the lungs Deficiency of vitamins C or P may be accompanied by lowered capillary resistance and by a tendency to spontaneous petechiæ and ecchymoses Lack of vitamin K may be associated with chronic cholecystitis jaundice and hypoprothrombinæmia and result in hæmoptysis from an old standing tuberculous cavity

**Differential Diagnosis** The diagnosis of hæmoptysis presents no difficulty if the patient is seen during an attack If there is only the history to help there may be great difficulty in differentiating it from hæmatemesis Attention should be paid to the following points Was the blood coughed up or vomited? Is there a history of previous chest trouble or of indigestion? Did the patient feel faint before the blood came up as often is the case with hæmatemesis Was there food mixed with the blood? Was the blood of a brownish colour due to the action of gastric juice or was it bright and frothy? Was there blood in the sputum for some days subsequently? Did melæna occur after the hæmorrhage? Melæna is not absolutely diagnostic of hæmatemesis as blood may be swallowed in a severe hæmoptysis If the blood is actually seen the reaction to litmus should be tested In hæmatemesis it is acid unless the patient has taken a large dose of alkali just before the bleeding took place whereas in hæmoptysis the reaction is alkaline In all cases a history of a definite hæmoptysis should be treated seriously and as soon as possible further investigations should be carried out to determine its cause In some cases however a cause cannot be found

**Treatment** In cases of pulmonary tuberculosis the patient should be propped up in bed in a semi recumbent position and if it is known from which lung the bleeding is coming he should be inclined slightly to that side This help to prevent the blood being aspirated into the healthy lung and carrying tuberculous infection there If the bleeding is severe or if the patient cannot be calmed a hypodermic injection of morphin sulph gr ¼ should be given immediately It is not wise to sit the patient up withhold morphine and encourage the patient to cough the blood up Such treatment may result in death The patient must keep still and not do anything for himself The chest may be auscultated but percussion must not be performed If the bleeding persists the morphine may be repeated but not more than a total of gr 1 should be given in 24 hours Bandages applied to the thighs with sufficient pressure to obstruct the venous but not the arterial circulation sometimes help to stop the hæmorrhage They should be kept on for half an hour and then can be applied to the upper arms for another half hour If the bleeding is very severe and threatening life an endeavour should be made to collapse the affected lung by means of an artificial pneumothorax Usually about 500 to 800 ml of air must be introduced into the pleural cavity to check the hæmorrhage If this is not possible owing to pleural adhesions artificial pneumoperitoneum is an alternative measure which has met with success It has the advantage that it is not necessary to know from which lung the bleeding is coming 700 to 800 ml of air are usually required to stop the bleeding refills being made twice in the first week and subsequently once a week for 6 to 8 weeks Cough should be allayed by a sedative linctus such as Tnc opii camph m 20 gly cerin m 20 and syr pruni serotin m 20 m 60 occasionally Alcohol ergot and digitalis should not be used All food should be taken cold but it need not necessarily be fluid thin bread and butter jellies cold fish

and cold chicken being allowed and a saline aperient of mag sulph gr 120 should be given Vitamin C deficiency can be diagnosed by studying the concentration of ascorbic acid in the urine before and after the administration of test doses of ascorbic acid If there is deficiency of vitamin C ascorbic acid tab mg 50 2 tabs t i d should be given If no response is obtained and the capillary resistance is lowered vitamin P should be administered, as Permidin tab 0 25 G, 4 tabs daily If the hæmoptysis is associated with hypoprothrombinæmia vitamin K should be prescribed, as Synkavit, one tab mg 10 t i d by mouth together with Bilein gr 5 capsule, 2 t i d by mouth if there is jaundice Alternatively Synkavit, mg 10 is injected intravenously or intramuscularly for 3 or 4 days

## Abscess of the Lungs

**Definition** *A localised purulent necrotising infection of the lungs*

**Etiology** The abscess is caused by infection with pyogenic organisms such as streptococci staphylococci pneumococci spirochætes or anaerobes Spirochætes are almost always present in the pus of inhalation abscesses The varieties of lung abscess may be classified as follows 1 *Aspiration* This is due to inhalation of septic material as after tonsillectomy or other operation on the naso pharynx Vomited material may be inhaled under anæsthesia A quinsy may rupture and the contents be inhaled during sleep Thirty seven per cent of all lung abscesses are of this inhalation type 2 *Bronchogenic* This is associated with foreign bodies in the bronchi tumours especially carcinoma bronchial stenosis and bronchitis Twenty five per cent of all lung abscesses are associated with bronchial carcinoma 3 *Hæmatogenous* The infecting organism is carried to the lungs in the blood stream Staphylococcal septicæmia is a frequent cause in children 4 *Necrotising pneumonia* This may occur after bronchopneumonia or lobar pneumonia usually due to a Friedlander s bacillus infection which may be combined with staphylococci Small multiple abscesses form which may become chronic 5 *Infected cysts* Bronchogenic cysts are often infected and may lead to abscess formation 6 *Transdiaphragmatic* Subphrenic abscess or amœbic abscess of the liver may give rise to pulmonary abscess formation 7 *Mediastinal* Suppuration in the mediastinum secondary to carcinoma of the œsophagus or suppuration in the bronchial glands may result in pulmonary abscess A lung hæmatoma may also suppurate The amœbic pulmonary abscess is described separately

**Pathology** *The inhalation abscess is usually in the right lung* especially in the axillary and posterior sub apical segments of the upper lobe less frequently in the apical segment of the lower lobe Embolic abscesses may be multiple and often they are close to the pleura The abscess may be encapsulated and closed or in connection with a bronchus

**Clinical Findings** Since the use of antibiotics the incidence of lung abscess has been considerably reduced A typical example of inhalation

abscess is as follows A day or so after the inhalation of infected material the temperature rises After a few days the patient notices that his breath is offensive after cough The temperature then rises higher and the patient is acutely ill

*On Examination* In the early stages a small area of dulness with diminished tactile fremitus weak breath sounds and a few râles is detected in one lung Unless the abscess is in connection with a bronchus there is no purulent sputum The temperature and pulse and respiration rates are all raised If the abscess ruptures into a bronchus offensive purulent sputum is produced This may measure about 14 oz or more in the 24 hours With the appearance of the pus the temperature usually falls and the general condition of the patient is ameliorated The blood shows a leucocytosis of about 12 000 to 15 000 per c mm The X rays reveal a shadow in the lung at first homogeneous but after rupture into a bronchus a cavity with a fluid level may be seen when the patient is in the erect position This may only be visible in a lateral radiogram The X ray appearance of staphylococcal abscesses has been described on p 154

**Differential Diagnosis** In the early stages a pulmonary abscess may be mistaken for bronchopneumonia or an encysted interlobar empyema After rupture has occurred the possibility of bronchiectasis must be considered The history of the case and the clinical and X ray findings usually render the diagnosis clear It should always be remembered that a lung abscess may be associated with a bronchial carcinoma the former being the prominent feature The diagnosis is generally established by bronchoscopy

**Course and Complications** In some cases the patient coughs up the contents of the abscess the temperature rapidly falls to normal and a spontaneous cure is effected In untreated cases the expectoration is continuously purulent and the temperature falls with periodical rises as retention of secretion occurs The suppurative process is slowly continuous recurrent hæmoptysis and eventually bronchiectasis may result The infection may be very acute or the patient s resistance very low and the lung then becomes gangrenous Metastatic foci may form elsewhere such as an abscess in the brain

**Prognosis** This has been much improved by antibiotic treatment

**Treatment** *Prophylactic* Acute pulmonary infections should be adequately treated by antibiotics Intramuscular injections of 1 million units of crystalline penicillin should be given daily for a week after operations on the nose and throat *Curative* One million units of crystalline penicillin should be injected intramuscularly every 12 hours combined with 0 5 G streptomycin every 6 hours Postural drainage should be used if the abscess is in a position to respond to such treatment and is in communication with a bronchus but this should be postponed until the acute stage is over If a cure is not effected in 2 or 3 weeks further measures will be required An attempt may be made to empty the abscess by passing a bronchoscope under local anæsthesia The operation of choice is now usually lobectomy rather than drainage through the chest wall especially in chronic abscess

### Gangrene of the Lungs

**Etiology** Gangrene of the lungs is now so rare owing to the early diagnosis and efficient treatment of the predisposing lung conditions that it hardly merits a separate description The sputum is horribly offensive and contains portions of disintegrated lung tissue and numerous aerobic and anaerobic organisms The patient is desperately ill with severe sweats and high fever, and the few cases I have seen have succumbed to the infection

### Hydatid Disease of the Lungs

**Etiology** The disease is due to infection with the ovum of the Echinococcus granulosus (Tænia echinococcus)

**Pathology** The cyst usually forms in a lower lobe of the lungs frequently on the right side It is generally solitary and surrounded by a fibrous capsule It may rupture into a bronchus, or into the pleura pericardium aorta pulmonary veins or through the diaphragm The cyst varies in size up to about 4 or 5 inches in diameter In some instances its contents inspissate

**Clinical Findings** The patient may give a history of residence in some country such as Australia where hydatid disease is comparatively common He may not notice any alteration in his health and the cyst is discovered by X ray examination In other cases the early symptoms are those of bronchitis or of an intra thoracic tumour Thus there may be cough with expectoration or progressive dyspnœa

*On Examination* The physical signs in the lungs may be very slight or a definite area of dulness may be detected in one lung with weak breath sounds and diminished voice conduction As the cyst enlarges the mediastinal contents are displaced towards the opposite side The blood may show an eosinophilia up to 6 or 8%, and the intradermal Casoni test (see p 84) is usually positive If the cyst ruptures into a bronchus hooklets may be found in the sputum

**Differential Diagnosis** The X ray findings are fairly characteristic the cystic shadow having a defined margin especially in the lateral view Other conditions such as a dermoid cyst or a simple pulmonary tumour may cause difficulties The blood and cutaneous tests for hydatid are useful for confirmatory evidence

**Course and Complications** A progressive enlargement of the cyst will give rise to increasing dyspnœa, whereas shrinking and inspissation are followed by a disappearance of all symptoms Rupture into a bronchus may be followed by a pulmonary abscess rupture into the pleura by shock and urticaria and rupture into the pericardium aorta or a pulmonary vein may cause sudden death

**Treatment** *The cyst should not be aspirated* Thoracotomy by a skilled surgeon will often enable the cyst to be removed.

### Dermoid Cysts of the Lungs

**Clinical Findings** There are usually no symptoms until the cyst enlarges sufficiently to cause pressure effects The patient may then

complain of cough expectoration shortness of breath or of pain in the chest Hairs may be seen in the sputum, and the presence of the cyst is revealed by X ray examination Instead of rupturing into the lung the dermoid may enlarge considerably compressing the lung into a thin layer The physical signs are then those of pleural effusion On aspiration through the chest wall the diagnosis may be established by finding hairs in the pultaceous contents of the cyst

**Treatment** If causing pressure effects an attempt at removal should be made by a skilled chest surgeon

## Congenital Cysts of the Lungs

**Etiology** Congenital cysts are uncommon and are probably formed by a dilatation of the atria of the lungs as no alveoli are present

**Pathology** In children or adults single or multiple cysts of varying size may be found containing air or fluid The fluid contents may be watery mucoid or purulent Congenital cysts are classified as 1 The large balloon cyst which may completely compress the lung in an infant or young child 2 The solitary cyst occupying up to half the lung field 3 Multiple medium sized cysts often seen near the lung root 4 Multiple small cysts resembling bronchiectasis They may be distinguished microscopically from bronchiectasis for with congenital cysts the cartilage muscle elastic tissue and glands are irregularly distributed in the supporting tissues the lining epithelium often remaining intact With bronchiectasis the epithelium is destroyed whereas the structures in the wall maintain their normal position

**Clinical Findings** An infant may be still born and at autopsy the lungs are full of small cysts In an infant or young child sudden dyspnœa may result from distension of a large cyst Congenital cystic disease in adults may give rise to recurring attacks of dyspnœa cyanosis and hæmoptysis When infection occurs there is loss of weight irregular fever cough offensive sputum and clubbing of the fingers This may be followed by lung abscess bronchiectasis empyema or cerebral abscess A cyst may rupture and cause a simple or benign spontaneous pneumothorax

**Differential Diagnosis** A single air containing cyst may be erroneously diagnosed as a cavity due to pulmonary tuberculosis or as a pneumothorax When infected the clinical findings resemble those of lung abscess or bronchiectasis

**Treatment** This varies with the type of cyst Pressure symptoms in a large balloon cyst must be immediately relieved by the insertion of a needle followed later by pneumonectomy or by removal of the cyst if it is pedunculated Radical cure of unilateral infected cysts may be effected by lobectomy or pneumonectomy

## Paragonomiasis

*(Pulmonary Distomiasis Lung Fluke Disease)*

**Etiology** The causative organism is a trematode the Paragonimus westermani or Distoma ringeri Infection in man occurs by drinking

infected water by bathing in it, or by eating infected crabs The disease is endemic in Japan, Formosa, Korea China and the Philippines

**Pathology** The trematode makes burrows in the lung

**Clinical Findings** The patient complains of recurrent hæmoptysis often related to exercise The diagnosis is established by finding the ova in the sputum

**Treatment** *Prophylactic* In infected areas all drinking water must be boiled bathing should be forbidden and crabs must not be eaten

*Curative* *This is as for bilharziasis*

## THE PLEURÆ

### Acute Dry Pleurisy

### (*Fibrinous or Plastic Pleurisy*)

**Definition** Inflammation of the pleura not accompanied by a fluid exudate

**Etiology** Primary cases may be due to a rheumatic infection following exposure to cold Cases described as acute benign dry pleurisy were met with in the Middle East amongst troops during the 1939–45 war Pleurisy is often secondary to a tuberculous focus in the lungs to *pneumonia pulmonary infarct bronchiectasis a neoplasm of the lungs* or to injury of the chest wall and at times to pulmonary actinomycosis

**Pathology** The inflammation may be localised or diffuse both layers of the pleura are usually involved, a sticky exudate of lymph and fibrin being found The pleurisy may be localised to an interlobar septum, or to the diaphragmatic pleura

**Clinical Findings** In the primary cases and in those associated with tuberculosis the patient is often a young adult who says that he was suddenly seized with a pain in the side resembling a ' stitch Any attempt at taking a deep breath or coughing intensifies the pain It should be remembered that in some cases there may be no pain although an extensive pleural rub is present

*On Examination* The temperature is usually a little raised 99 or 100° F The respiration rate is also somewhat above the normal but the breathing is shallow The patient may prefer to lie in bed on his back or on the sound side owing to local tenderness over the affected area

The chest Inspection The movement may be slightly diminished on the affected side Palpation Often there is local tenderness at the site of the pain Friction fremitus may be felt Percussion *The resonance is normal or slightly diminished* Auscultation There is weak air entry over the area of the pleurisy A leathery or creaking rub may be heard at the end of inspiration or beginning of expiration or fine pleural crepitations at the end of inspiration These are constant after cough helping to differentiate them from pulmonary rales which often disappear after cough Vocal resonance may be slightly diminished over the affected area

In diaphragmatic pleurisy *the pain is often referred to the tip of*

the corresponding shoulder or to the shoulder joint (the reflex is through the phrenic nerve to the 4th cervical nerve root) or the symptoms may be abdominal In the latter case a tender area can be found in the subcostal plane about 2 inches from the mid line (*le bouton de Guéneau de Mussey*) There is usually weak air entry over the corresponding lower lobe of the lung In interlobar pleurisy the inflammation may be localised to the septum between the right upper and middle lobes The pain is then felt in the region of the fourth right costal cartilage and fine crepitations may be audible at this level Often the condition is only revealed by X ray examination

**Differential Diagnosis** Other causes of pain in the chest must be considered such as myalgia or fibrositis of the intercostal muscles or membranes (pleurodynia) Bornholm disease intercostal neuralgia or neuritis periostitis of a rib the initial pain of herpes zoster and pain referred from the heart or abdomen The physical signs of dry pleurisy enumerated above are very characteristic In intercostal myalgia although the pain is intensified by deep breathing no pleural signs are found In intercostal neuritis or neuralgia the pain follows the nerve path and localised tender spots are present especially at the back (posterior primary division) in the mid axilla (lateral cutaneous branch) and at the chondro sternal junction (anterior cutaneous branch) The appearance of the typical eruption establishes the diagnosis of herpes Rib periostitis may be shown by X ray examination Pain due to cardiac or gastric causes is often related to effort or to digestion

**Course and Complications** In simple cases the pain usually lasts for about a week but fluid may rapidly form and by separating the inflamed surfaces cause the pain to disappear In other cases the pain of dry pleurisy is rapidly followed by the symptoms and signs of lobar pneumonia or it may be the initial symptom of a pulmonary neoplasm or of tuberculosis An extensive plastic pleurisy may pass on to pleurogenous fibrosis of the lungs

**Prognosis** This depends largely upon the cause The possibility of pulmonary tuberculosis or of a neoplasm should always be borne in mind

**Treatment** The patient must be kept in bed until the temperature is normal The pain may sometimes be relieved by the application of liq iodi mitis or cataplasma kaolini to the affected side If this is unsuccessful the side should be strapped from base to apex in a position of full expiration The useless dry cough can be checked by a sedative linctus such as Oxymel scillæ m 30 acid hydrocyan dil m 2 morphin tartrat gr 1/16 aquam ad m 60 To secure sleep some drug such as aspirin gr 5 to 10 may be given at night The diet during the febrile period should be fluid and the bowels should be opened daily with salines such as mag sulph gr 60 to 120 mane When the temperature has fallen the chest should be X rayed to see if there is any evidence of underlying pulmonary disease If a tuberculous focus is discovered the treatment is as for an early case of pulmonary tuberculosis Apart from this gentle breathing exercises should be encouraged during

convalescence to expand the lung increase the circulation and preven the formation of pulmonary fibrosis

### Chronic Dry Pleurisy

Chronic thickening of the pleura sometimes with calcified plaques may be found in association with pulmonary *tuberculosis in artificia* pneumothorax or pleural effusions of some duration after an empyema and in polyserositis

### Pleural Effusion

The following varieties are described 1 Clear effusions of which there are two varieties, pleurisy with effusion (erothorax) and a pleural transudate (hydrothorax) 2 Purulent effusion an empyema (pyothorax) 3 *Hæmorrhagic effusions,* such as hæmothorax hæmo serothorax, and hæmohydrothorax 4 Milky effusions such as chylothorax pseudochylothorax and chyliform effusions

### Pleurisy with Effusion

*(Sero fibrinous pleurisy Serothorax)*

**Definition** An inflammatory serous pleural exudate

**Etiology** *The most important cause is a tuberculous focus in the* lungs or pleura Some cases may be due to rheumatism or occur as a complication of septicæmia or enteric fever Less commonly the effusion is associated with inflammatory pulmonary lesions such as pneumonia virus pneumonia and bronchopneumonia with a pulmonary *infarct or with malignant disease of the lungs pleura mediastinum or* ribs It may also be met with as a complication of lesions of the peri cardium or abdomen, or as a manifestation of polyserositis Artificial pneumothorax is complicated by a serous effusion in about 50% of cases

**Pathology** A plastic pleurisy is followed by the output of a clear *yellow exudate The specific gravity of the fluid is between 1 010* and 1 018 The reaction is alkaline, and protein (albumin globulin and fibrinogen) is present to the extent of 4% or over The fluid usually clots on standing Microscopically polymorphonuclear cells lymphocytes and a few red cells are seen and occasionally eosinophils *predominate In one case with combined abdominal tuberculosis* and a tuberculous pleural effusion the fluid showed 80% and the blood 14% of eosinophils A few pleural endothelial cells may also be seen The exudate is often sterile but tubercle bacilli may be found or if the exudate is injected into a guinea pig it may cause the animal s death *from tuberculosis Lymphocytosis is suggestive of a* tuberculous infection In other cases the pneumococcus or Bact typhosum may be found The amount of fluid varies from a few ounces to over 2 pints

**Clinical Findings** The effusion may arise insidiously or follow an attack of acute dry pleurisy, or occur as a complication of one of the *conditions mentioned above* The symptoms vary with the mode of onset and the patient may complain of pain or of dyspnœa The results produced by the effusion depend largely upon the amount of fluid present

*On Examination:* In an average case there is fever of *101° F or*

over, the pulse is frequent 100 or more and the respiration rate is above normal The patient appears distressed on slight exertion and there is some cyanosis He tends to lie on the affected side or may require to be propped up The chest Inspection There is diminished movement on the affected side The shadow of the diaphragmatic movement as seen on the chest wall when the patient breathes is not visible on the affected side (Litten s sign) The cardiac impulse may be seen displaced away from the side of the effusion Palpation There is diminished expansion on the affected side Tactile fremitus is usually absent over the effusion The cardiac apex is moved away from the side of the fluid and in a left sided effusion cardiac pulsation may be felt to the right of the sternum Percussion There is stony dulness over the effusion above the level of the fluid a high pitched note may be elicited (skodaic resonance) due to compression of the lung The line of the upper level of the fluid in a moderate sized effusion is curved from before backwards the highest point being situated in the posterior part of the axilla (the S curve of Ellis or Damoiseau s line) The level of the upper border of the fluid may also vary with change of position of the patient A triangular area of dulness may be found on the opposite side at the back owing to the fluid forming a mediastinal bulge (Grocco s triangle) The apex of this triangle is on top near the spine at the level of the upper border of the effusion Auscultation The breath sounds over the fluid may be absent very weak or bronchial in character if the lung is collapsed and the bronchi remain patent The air entry is usually feeble above the fluid whereas over the opposite lung the breath sounds are loud and harsh Some scattered rhonchi may be heard above the fluid and a few râles at the opposite base Usually there are no adventitious sounds over the dull area but at times some coarse rales are audible A pleural friction rub may be heard just above the upper fluid level Vocal resonance is usually absent over the fluid but near its upper level the conducted voice sound has a nasal or bleating character (ægophony) Whispering pectoriloquy and broncho phony are occasionally present over a pleural effusion A large pleural effusion may cause a downward displacement of either the liver or the spleen

**Differential Diagnosis** The nature of the fluid in the chest can only be determined by exploratory puncture The diagnosis of the presence of a pleural effusion is often a matter of great difficulty especially if the fluid is of small volume or loculated by pleural adhesions The most reliable indications of fluid are the triad of signs stony dulness absence of tactile fremitus and absence of adventitious sounds Cardiac displacement is not always present The breath sounds are so variable that no reliance can be placed on them A pleural effusion must be differentiated from the following conditions Thickened pleura and fibrosis of lung consolidation of the lung and pericardial effusion With a thickened pleura and fibroid lung the dulness is not of such a stony character the chest wall is often flattened and the heart may be displaced towards the affected side Adventitious sounds such as fibroid rales are often heard over the affected lobe In pulmonary

consolidation the note also is not so stony, vocal fremitus is increased, and rales are usually audible There is also no cardiac displacement *In pericardial effusion* the dulness has a peculiar shape (see p 217) and the signs at the left base behind are those of consolidation (see p 217) In all cases of doubt especially in a localised interlobar mediastinal or *diaphragmatic effusion* an X ray examination is of great value, and should be obtained, if possible before any exploratory puncture of the chest is made

**Course and Complications** A small pleural effusion is usually absorbed spontaneously in 2 to 3 weeks with some resultant pleural thickening adhesions or calcification The compressed lung re expands and very little abnormality can be detected subsequently In other cases the lower portion of the lung does not completely expand is con gested, and fibrosis of the lung ensues Acute œdema of the lungs is a rare complication during the early stage of the disease A large effusion often will not absorb until a certain amount has been removed by aspiration In other cases after aspiration the effusion recurs repeatedly causing little constitutional or mechanical disturbance A thick layer of fibrin may be deposited on the pleura constituting a fibrothorax

**Prognosis** The immediate prognosis is good, but in about 30% of cases of pleural effusion forming between the ages of 15 and 40 years pulmonary tuberculosis develops within 5 years Less frequently bronchiectasis ensues on fibrosis of the lung

**Treatment** The patient should be kept in bed as long as the temperature is raised, and if practicable until the effusion is absorbed The amount of fluid ingested should be limited to 2 pints a day, and in obstinate cases a salt poor diet (see p 474) may be given The affected part of the chest should be painted every other day with *liq iodi mit* A syringe full of fluid should be removed from the pleura and examined chemically cytologically and bacteriologically *Aspiration with air replacement should be performed under the following circum stances* 1 If the fluid reaches to the second rib in the erect posture 2 If there is marked dyspnœa or cardiac embarrassment 3 If the fluid does not show signs of absorption in 3 weeks 4 If there is a bilateral effusion the larger exudate should be aspirated 5 If acute œdema of the lungs or severe collateral hyperæmia of the lungs occurs 6 If X ray examination reveals a tuberculous focus in the lung or if tubercle bacilli are found in the sputum It is not advisable to aspirate all pleural effusions as a routine procedure After the fluid has absorbed the lungs should be X rayed again and if no evidence of tuberculosis is obtained breathing exercises should be instituted to expand the lung and prevent the occurrence of fibrosis and of bronchiectasis The sound side of the chest should be prevented from expanding by means of a towel held round it or by the back of a chair pressed up against it, whilst inspiratory efforts are made for 5 minutes night and morning If this exercise causes persistent pain or aching in the chest it should be discontinued for a time In all cases in which there is evidence of tuberculosis a course of streptomycin I N A H and/or P A S is advisable (see p 168)

## Hydrothorax

**Definition** A transudate into the pleural space

**Etiology** The two main causes of hydrothorax are heart failure and renal disease More rarely it is associated with severe anæmia famine œdema or deficiency of the vitamin B complex or with thrombosis of the azygos veins or pressure upon them by a mediastinal growth or by mediastinal glandular enlargement due to Hodgkin s disease or leukæmia Meigs syndrome first described by Cullingworth in London in 1879 and later in America by Meigs in 1934 is characterised by an ovarian fibroma with ascites and hydrothorax It is suggested that the hydrothorax results from mechanical obstruction to the venous return within the abdomen or that the fluid passes by lymphatics through the diaphragm to the pleura or through a persistent pleuro peritoneal canal Hydrothorax and ascites occasionally occur associated with cirrhosis of the liver

**Pathology** The fluid transudes either as the result of venous stasis or owing to changes in the blood and tissues associated with renal disease The transudate is pale yellow the specific gravity is lower than 1 010 and a small quantity of protein (albumin and globulin) is present This is less than 3% It does not clot on standing Microscopically only a few endothelial cells are present and there are no organisms The transudate may be bilateral

**Clinical Findings** The history is that of the antecedent condition usually cardiac or renal disease An increase of dyspnœa suggests fluid in the pleura the physical signs of which have been described above Cardiac displacement is not generally marked There is no disturbance of temperature and no preliminary stage of dry pleurisy is noted Œdema is often present elsewhere in the legs hands or back Cases of Meigs syndrome are likely to be considered to be malignant and inoperable and so it is important that a correct diagnosis is made The pleural effusion is large unilateral generally on the right side and displaces the mediastinum The ovarian fibroma can usually be removed with subsequent cure of the condition

**Course and Complications** The course depends largely upon the results of the treatment employed for the underlying cause

**Prognosis** This is often grave as in cardiac disease the transudate is an indication of circulatory failure and in renal disease it is an expression of defective elimination

**Treatment** The fluid should be aspirated if it is causing distress and the appropriate treatment given for the œdema of cardiac or renal disease or for vitamin B deficiency

## Empyema

(*Pyothorax*)

**Definition** Pus in the pleural cavity

**Etiology** The incidence of empyema has been much lowered by the use of sulphonamides and antibiotics and it is now a comparatively rare disease Empyema was formerly most often a complication of

lobar pneumonia or of bronchopneumonia It may be syn pneun or meta pneumonic, according to whether it develops during the febrile stage of pneumonia or as a complication after the temper has fallen to normal Other causes are pulmonary tubercu bronchiectasis abscess or gangrene of the lung mediastinal abs carcinoma of the œsophagus pericarditis penetrating wounds of chest, subphrenic abscess septicæmia and pyæmia Rarely it app to develop as a primary condition The causative organisms inc the Diplococcus pneumoniæ (pneumococcus) streptococci the M bacterium tuberculosis (B tuberculosis) the Bacterium comm (B coli) the Hæmophilus influenzæ the Bacterium friedlanderi (pneu bacillus) the Bacterium typhosum (B typhosus) staphylococci varieties of streptothrix A sterile purulent effusion is met wit tuberculous pyothorax, and in some acute cases for a day or so the rupture of a lung abscess

**Pathology** The pleura becomes thickened and the lung colla and fibrosed in long standing cases The pleural space becomes abscess cavity, and the walls a pyogenic membrane, which is o resistant to antibiotic treatment necessitating excision The fluid be odourless or very offensive, it may be thin or thick, yellow greenish or brown Large fibrinous flakes are often present in pneu coccal infections whereas in a streptococcal empyema the fluid is o thin The specific gravity is usually over 1 030 and numerous di tegrating pus cells are present The fluid may be free or loculate the base apex, on the mediastinal surface or in an interlobar fiss It may track through the chest wall into the lungs or pericardium through the diaphragm

**Clinical Findings** Empyema is chiefly a disease of children young adults although it may occur at any age In a typical me pneumonic case after an attack of lobar pneumonia, the temperatu which has fallen to normal for a few days, gradually rises again irregular stages The condition of the patient deteriorates the appe fails and shivering or sweating attacks may occur There may be actual rigor As the effusion increases in size the toxæmic sympto are more marked the pulse and respiration rates increase and there dyspnœa

*On Examination* Often there are no special symptoms T physical signs of pleural effusion may be found but they are oft localised to a small area In addition the special features met with empyema are as follows The patient is often pale and has a toxæn appearance There may be bulging of the intercostal spaces on t affected side or œdema of the chest wall or corresponding arm Pulsati may be seen in left sided cases usually near the apex of the he (*empyema necessitatis*) when the empyema tracks through the int costal muscles Clubbing of the fingers may rapidly appear T breath or sputum is offensive if the empyema is interlobar a communicates with a bronchus There is usually a leucocytosis between 15 000 and 20 000 per c mm X ray examination is of value

**Differential Diagnosis** It is easy to overlook an empyema especially when the onset is insidious, as so often is the case in pneumonia It should always be suspected if the temperature rises again after it has fallen to normal unless this rise can be accounted for by some other complication such as spread of the disease or the development of pericarditis In all cases of doubt an exploratory puncture should be made, but in some cases the pus is thick and will only pass through a wide bore needle An apical empyema is a source of difficulty it may be mistaken for a pulmonary neoplasm but in empyema the dulness does not extend across the mid sternal line as it may do with a new growth An interlobar empyema is to be suspected when localised signs of fluid are discovered along the line of an interlobar septum or if the breath is offensive on coughing The X rays are of inestimable value in the diagnosis The following conditions also must be excluded in certain cases Unresolved pneumonia and fibrosis of the lung tuberculosis or a growth of the lung a rib abscess an aneurysm and a subphrenic abscess In unresolved pneumonia and pulmonary fibrosis the heart is not displaced away from the affected side and usually rales are heard over the dull area and tactile fremitus is present In doubtful cases exploratory puncture should be made A rib abscess may be mistaken for a pointing empyema or an aneurysm for a pulsating empyema The X ray examination is invaluable in such cases In subphrenic abscess help is obtained from the history of the antecedent condition and the X ray examination which may reveal the situation of the diaphragm raised in a subphrenic abscess and depressed in an empyema There is usually no lateral cardiac displacement with a subphrenic abscess If an exploratory needle is inserted through a lower intercostal space and passed downwards until the pus is located the needle will move with respiration in a subphrenic collection as it must pierce the diaphragm

**Course and Complications** Unless the empyema is effectively treated the patient usually becomes more severely ill and will eventually die from heart failure pyæmia amyloid disease or cerebral abscess In rare instances the virulence of the empyema abates and a sterile empyema results with calcification of the pleura Further, an empyema may develop on the opposite side or the pus may track through the chest wall into the mediastinum pericardium lung or into the neck or abdomen If an empyema ruptures into a bronchus it may result in spontaneous cure or form a bronchial fistula or cause death by suffocation Other complications include abscess or gangrene of the lungs bronchiectasis pulmonary osteo arthropathy pyæmia and abscess in skeletal muscles Permanent pleural thickening or fibrothorax usually remains as a sequela of an empyema

**Prognosis** Empyema is a serious condition but the prognosis is naturally improved by early diagnosis and efficient treatment Unless adequately treated there is a danger of the empyema becoming chronic

**Treatment** In all cases of doubt the chest should be explored In loculated or interlobar empyema it may be necessary to perform several punctures under a general anæsthetic the surgeon being then

prepared to proceed to evacuate the pus if it is located Penicillin s be administered intrapleurally following aspiration of the pleural flu the treatment of empyema due to pneumococci streptococci or stap cocci but not in cases of Bact coli infection The amount inj is usually 1 million units in 10 ml sterile water Three or four aspira and instillations on alternate days generally suffice A specimen o should be withdrawn the day after each aspiration to see if orga still persist For hæmolytic staphylococcus aureus and Bact infections aureomycin mg 100 or chloramphenicol mg 250 in *sterile saline solution should be introduced into the pleura inste* penicillin Thoracotomy was formerly required to evacuate blood fibrin and pus but the introduction into the pleural cavity of 20 units of streptokinase and *75 000 units of streptodornase may* this necessity These substances cause lysis of the clots There remain some cases which require surgical drainage or more exte operations on the lung and pleura Local penicillin treatment rib resection is often unsatisfactory and crystalline penicillin is best given by intramuscular injections of 1 million units every 12 h After treatment consists in the use of suitable inspiratory breat exercises to expand the lung A tuberculous empyema should neve treated at first by rib resection but the fluid should be repeat aspirated injecting 1 G of streptomycin into the pleural cavity *each aspiration A course of streptomycin should also be given* intramuscular injections and I N A H administered by mouth some cases thoracoplasty or extrapleural pneumonectomy is necess

### Hæmothorax

**Definition** Blood in the pleural cavity

**Etiology** Hæmothorax may result from wounds of the chest or the lungs, from blast of high explosives, from fractured ribs inju the lung from division of pleural adhesions, or from rupture of aneurysm *Hæmorrhage into aberrant lung tissue may rupture* the pleura It has also been recorded on the left side due to a stran lated diaphragmatic hernia containing omentum and gangren stomach

**Pathology** In gunshot wounds of the chest the blood pours into the pleural space and does not clot The diaphragm is usua displaced upwards on the affected side and the lung is collapsed T hæmothorax may become infected with streptococci or with anaerob In other cases death rapidly ensues and the blood is clotted

**Clinical Findings** The patient who has had a penetrating wou *of the chest usually gives a history of hæmoptysis following* directly the wound He then complains of pain in the chest cough and dyspn

*On Examination* The signs of a pleural effusion are found the fluid is sterile there is little or no pyrexia but if as is often t case it is infected a high temperature is registered In additi as air is often present in the pleural cavity together with blood (trauma hæmopneumothorax) the signs of fluid and air may also be detected

**Differential Diagnosis** The nature of the fluid is determined by exploratory puncture

**Course and Complications** The course is usually that of an uncomplicated pleural effusion with gradual absorption of the fluid If the blood becomes infected the temperature suddenly rises to 102 F or higher and the patient becomes desperately ill

**Prognosis** This is usually good in cases due to wounds If the hæmothorax is due to rupture of an aneurysm death occurs in a few minutes

**Treatment** Aspiration of the fluid aids absorption but this is not required in small effusions Removal of air from the pleural cavity may be required if the pressure is high In all cases a course of intramuscular penicillin should be given If the fluid is infected the treatment is the same as for empyema

### Hæmoserothorax

**Definition** An inflammatory serous pleural exudate containing blood

**Etiology** Hæmoserothorax may be met with in association with new growths of the lung pleura or mediastinum with pulmonary or pleural tuberculosis or after previous aspirations of a serothorax It usually follows pneumonectomy At times it is a complication of cirrhosis of the liver scarlet fever small pox purpura or lobar pneumonia

No special description of the clinical findings is called for as they resemble those of sero fibrinous pleurisy The finding of blood in the pleural fluid should always be regarded as a very suspicious indication of an intra thoracic new growth

### Hæmohydrothorax

**Definition** A pleural transudate containing blood

**Etiology** Hæmohydrothorax is most usually associated with heart failure The clinical findings are those of hydrothorax

### Chylothorax

**Definition** A pleural effusion of chyle or of fluid containing chyle

**Etiology** Chylothorax results from injury of or obstruction to the thoracic duct These may be due to trauma erosion by a growth pressure of a growth or of enlarged mediastinal glands or to internal blockage by a parasite such as the Wucheria bancrofti

**Pathology** The fluid is milky and contains fat On standing the fat forms a layer on top of the fluid

**Clinical Findings** No special description is called for The nature of the fluid is discovered by thoracic puncture The fluid should not be aspirated unless causing cardiac embarrassment or dyspnœa

### Pseudochylothorax

**Definition** An opalescent pleural effusion which does not contain fat globules

**Etiology** Pseudochylothorax may be associated with chronic pulmonary tuberculosis malignant disease of the lungs or pleura and chronic disease of the heart or kidneys

**Pathology** The opalescence of the fluid may be caused by the presence of lecithin globulin calcium phosphate cholesterin or filariæ On standing a deposit forms, which in the case of cholesterin effusions, is seen as delicate silvery flakes

**Clinical Findings** The signs are those of a pleural effusion The nature of the fluid is determined by removing a sample Aspiration is rarely required

## Chyliform Effusions

**Definition** A pleural effusion containing fat globules but no chyle

**Etiology** Chyliform effusions may be associated with tuberculosis or malignant disease of the lungs or pleura

**Pathology** The fluid has a milky appearance and remains turbid on standing The fat globules are probably derived from degenerating leucocytes and endothelial cells

**Clinical Findings** A sample of fluid should be removed for diagnostic examination

## Pneumothorax

(*including Hydropneumothorax and Pyopneumothorax*)

**Definition** Air or gas in the pleural cavity Serous fluid (hydropneumothorax) or less often pus (pyopneumothorax) may also be present

**Etiology** The air or gas may be derived from various sources 1 The lungs Rupture of a tuberculous focus accounts for many cases It may also complicate an artificial pneumothorax Less frequently the pulmonary lesion, which causes pneumothorax by its rupture, is an abscess gangrene new growth anthrasilicosis infarct or a hydatid cyst of the lungs or the perforation is caused by rupture of an empyema into the lung a fractured rib or an exploring needle ' Benign spontaneous pneumothorax, also called ' pneumothorax in the apparently healthy or pneumothorax simplex " constitutes a special group of cases Over 80% of the patients are males It is probably due to the rupture of an emphysematous bulla or congenital cyst of the lungs 2 The exterior The air may be introduced therapeutically as in artificial pneumothorax, or accidentally owing to a wound of the chest wall opening the pleura or during aspiration of a pleural effusion 3 The mediastinum The air may come from the œsophagus due to rupture This may be secondary to carcinoma to corrosive poisons or to trauma from an œsophagoscope or bougie or no cause may be found It may also follow bronchoscopy 4 The abdomen Pneumoperitoneum induction may cause pneumothorax because of a congenital pleuro peritoneal hiatus Perforation of the stomach duodenum or colon may lead to a subphrenic abscess which tracks through the diaphragm into the pleura A liver abscess may

rupture into the lung 5 The pleura Anaerobic organisms may form gas in a pleural effusion

**Pathology** In perforation of the lung the opening may remain patent air passing in and out with respiration or it may immediately seal over (closed pneumothorax) or it may form a valve air entering the pleural cavity with each inspiration and being unable to leave it with expiration This latter variety is known as suffocative pneumo thorax A bilateral pneumothorax may occur due either to rupture of both lungs or to the air escaping from one pneumothorax, through a minute opening into the opposite pleural cavity

**Clinical Findings** A pneumothorax may occur suddenly in a patient who is apparently in good health or as a complication of an illness which confines him to bed This illness is usually pulmonary tuberculosis The onset may occur while the patient is walking along the street or be associated with a cough or some muscular effort There is typically a sudden severe pain in the chest often in the mid axilla and a sensation of something snapping may be felt The patient may immediately collapse or find great distress in breathing The degree of distress depends upon the nature of the opening into the pleura whether valve like or otherwise and also upon the presence or absence of pleural adhesions which loculate the pneumothorax and prevent mediastinal displacement In the latter case the onset of the pneumothorax is often symptomless and it is only discovered on a routine examination

*On Examination* With a diffuse pneumothorax the patient is usually propped up in bed very distressed and short of breath The chest Inspection There is diminished movement on the affected side The cardiac impulse may be seen displaced towards the sound side Palpation Tactile fremitus is absent on the affected side The cardiac impulse is felt displaced away from the side of the pneumo thorax Percussion There is a hyperresonant note over the pneumothorax If fluid is also present the note is impaired at the base and shifting dulness may be obtained when the chest is percussed with the patient at first erect and then reclining The upper border of liver dulness may be obscured in right sided cases Auscultation The breath sounds over the pneumothorax are weak and may be of a metallic bronchial character The coin sound is often heard This can be elicited in a modified form by flicking the chest wall over the pneumo thorax with the finger nail while listening through the stethoscope Harsh breath sounds are audible over the other lung With a shallow left sided pneumothorax systolic clicks which are audible both to the patient and to the examiner may be heard near the apex of the heart Metallic râles or a splash (Hippocratic succussion) may be heard if fluid and air are present in the pleural space (hydropneumothorax) The pulse rate is usually about 120 and the respirations 20 to 30 The *diagnosis is confirmed by removal* of a small quantity of air by explora tory puncture A small syringe is used containing a 2% solution of procaine When the needle enters the pleural space bubbles of air can be sucked back into the syringe The X ray examination will

show a clear area due to the air and a collapsed lung If there is a hydropneumothorax the upper level of the fluid assumes a straight transverse line, when the patient is erect The physical signs in a loculated pneumothorax may closely resemble those of a pulmonary cavity, and X ray examination is of value in differentiating them

**Differential Diagnosis** There is usually no difficulty in the diagnosis of a complete spontaneous pneumothorax A loculated pneumothorax may simulate a pulmonary cavity or an emphysematous bulla Further a loculated apical pneumothorax, occurring in a patient who has extensive fibroid tuberculosis of the opposite lung, may cause sudden severe dyspnœa so that it is mistaken for asthma A partial left sided pneumothorax may symptomatically closely resemble coronary disease Careful exploratory puncture of the pleura as described above will demonstrate the presence of air If a pneumothorax needle is now inserted attached to a manometer it can be shown that the air is in the pleural sac and not in the lung In the former the mean pressure is positive whereas if the needle is in the lung there is a respiratory excursion of pressure above and below the zero line the mean pressure being zero A loculated basal pneumothorax may closely simulate a perforated gastric or duodenal ulcer but with a careful examination on the lines described above the diagnosis can usually be made

**Course and Complications** The course of a spontaneous pneumothorax depends upon its cause and the nature of the opening Recurrent attacks may occur or even bilateral pneumothorax The occurrence of an infected pleural effusion increases the gravity of the situation

**Prognosis** This is often grave death may rapidly occur from shock or the air may gradually be absorbed Bilateral cases are also of greater severity In advanced cases of pulmonary tuberculosis a spontaneous pneumothorax usually ushers in the final scene whereas in early cases or in 'simple pneumothorax' due to rupture of an emphysematous bulla, the outlook is usually good although recurrence occurs in about 20% of cases

**Treatment** A hypodermic injection of morphin sulph gr ¼ should be given immediately if the patient is in distress If the pressure of air is causing dyspnœa and cardiac embarrassment it must be reduced *This can best be done with the aid of a pneumothorax apparatus* If the needle is now kept in the pleural cavity for 5 or 10 minutes it can be seen if the pressure is rising again If this is occurring it will be necessary either to remove air frequently or to keep in the pleura a needle to which is attached a rubber tube connected by a T glass tube both with a pneumothorax apparatus to record the intrapleural pressure and with a tube the distal end of which dips under a disinfectant fluid such as 1/1 000 perchloride of mercury in a bottle on the floor A cannula with a blunt edge and not a sharp pointed needle should be left in the chest, as there is a risk of penetration of the lung and death from hæmoptysis If the lung fails to expand after 3 or 4 weeks an obliterative pleurisy may be induced by the injection of an irritant such as 5 ml of 1% camphor in olive oil

If a hydropneumothorax is present aspiration of the fluid will reduce the intrapleural pressure and so relieve distress A pyopneumothorax requires treatment as for empyema

### Spontaneous Hæmopneumothorax

This is a rare condition in which air and blood escape spontaneously into the pleural cavity In some cases the cause is a ruptured emphysematous bulla or a torn pleural adhesion in others no source of the bleeding or air leak is discovered at autopsy The treatment is as for spontaneous pneumothorax the blood also being aspirated from the pleural sac Transfusion of whole blood may be required to combat the anæmia and if the bleeding persists thoracotomy may be necessary

### New Growths of the Pleura

Simple tumours are rare They include a fibroma lipoma and angioma

Malignant growths may be primary such as an endothelioma carcinoma and sarcoma or secondary such as a carcinoma sarcoma or endometrioma

### Endothelioma of the Pleura

**Pathology** The growth spreads over the pleura and may cause considerable thickening of both layers It may also infiltrate the pericardium and heart and extend through the diaphragm the peritoneum being studded with minute nodules The two layers of the pleura may be studded with nodular growths up to 2½ inches in diameter without general pleural thickening A blood stained pleural effusion is often present The cells are chiefly endothelial They may be agminated in plaques or rosettes and show budding and mitotic changes (Foulis cells)

**Clinical Findings** The patient is usually an adult of middle age who complains of progressive loss of strength with cough and shortness of breath

*On Examination* The patient may be wasted and signs of a pleural effusion are present On exploring the chest it will be noticed that the needle passes through a considerably thickened pleura The fluid is usually blood stained as described above In some cases enlarged glands are found above the clavicles or in the axillæ and subcutaneous nodules may be felt along the ribs Air replacement of the fluid and subsequent thoracoscopic examination are of value in establishing the diagnosis in an early case when the area of growth can be visualised

**Course and Complications** The course is usually rapid Complications include extension to other parts of the body such as the heart and peritoneum

**Prognosis** Death usually occurs in 2 to 3 months from the date of diagnosis

**Treatment** This is usually only palliative In some early cases surgical removal is possible Dyspnœa can be relieved by aspiration

of the fluid Pain and cough can be treated only by sedatives such as morphin sulph gr $\frac{1}{8}$ to $\frac{1}{4}$

### Calcification of the Pleura

This may be demonstrated radiographically in certain long standing cases of encysted empyema or following a serous pleural effusion or chronic hæmothorax

## THE MEDIASTINUM

### Mediastinitis

**Definition** Inflammation of the mediastinal connective tissue

**Etiology** Acute mediastinitis is usually secondary to acute inflammation of the lungs or pericardium to rupture of the œsophagus or to wounds of the chest wall Syphilitic mediastinitis may cause obstruction of the superior vena cava

**Clinical Findings** The history and the examination of the patient usually suggest the primary cause The patient may complain of acute pain under the sternum and in the interscapular region If an abscess forms it may cause mediastinal pressure and it may point behind in the interscapular area

**Treatment** An abscess should be drained surgically

### Mediastinal Emphysema

**Definition** Air in the mediastinal connective tissue spaces

**Etiology** Mediastinal emphysema may result from rupture of the trachea, bronchi, or œsophagus or be secondary to acute interstitial emphysema of the lungs

**Clinical Findings** The symptoms of mediastinal emphysema are usually masked by those of the primary cause The patient may complain of pain under the sternum and, on examination a hyper resonant note is found over the sternum The heart sounds are distant and surgical emphysema may be detected in the neck

**Treatment** There is no special treatment apart from that required for the primary condition

### Enlarged Mediastinal Glands

**Etiology** The glands which lie in the posterior mediastinum may be enlarged from various causes such as 1 Simple inflammation in whooping cough measles glandular fever bronchopneumonia pneumonia pneumokoniosis and influenza 2 Granulomatous infection in tuberculosis sarcoidosis and syphilis 3 Hæmopoietic diseases such as Hodgkin s disease and leukæmia 4 Malignant disease such as sarcoma or carcinoma

### Tumours of the Mediastinum

**Varieties** *Simple tumours* are rare A lipoma may fill the anterior mediastinum Other simple tumours include a myoma neurofibroma chondroma osteochondroma persistent thymus and retrosternal goitre

*Malignant tumours* may be carcinoma sarcoma or ganglio-neuroblastoma Carcinoma probably is always secondary to a growth in the bronchi or lungs and some of the mediastinal sarcomata formerly described are probably of the nature of oat-celled carcinoma of the bronchus Lymphosarcoma Hodgkin s disease or leukæmia may also affect the mediastinal glands

**Clinical Findings** The special features of new growth of the mediastinum are the pressure effects which are known as *the mediastinal syndrome* Certain structures may be compressed The trachea or bronchi There is cough dyspnœa or stridor and collapse of the lung The nerves The vagus recurrent laryngeal sympathetic phrenic or intercostals may be affected Pressure may result in slowing or acceleration of the pulse hoarseness inequality of the pupils hiccough paradoxical movement of one side of the diaphragm (up with inspiration and down with expiration) and pain in the chest or arm The arteries The pulses may be unequal or the blood supply to one lung may be interfered with and gangrene ensue. The veins The superficial veins of the chest may dilate (see Fig 47) The superior vena cava may be obstructed with reversal of flow in the veins in the chest wall the current being now from above downwards in the upper part of the chest Œdema may also be seen in the face neck chest or arm the lungs may be hyperæmic from back pressure or a pleural effusion may develop The lungs A portion of the lungs may collapse The œsophagus Pressure will result in dysphagia The thoracic duct A chylous pleural effusion may develop The early symptoms are thus very variable a persistent ineffective cough may be complained of or pain in the chest or arm may be first noted The symptoms and signs are usually indistinguishable from those of new growth of the lungs The diagnosis is aided by the X ray examination and the determination of the Wassermann reaction which helps in the exclusion of an aneurysm

**Treatment.** This is purely palliative in all malignant cases as the prognosis is hopeless A simple tumour such as a lipoma of the anterior mediastinum or a retrosternal goitre can frequently be removed surgically X ray treatment should be given in cases of Hodgkin s disease leukæmia or sarcoma The immediate response in sarcoma or Hodgkin s disease is often good but recurrence of the growth inevitably occurs

### Cysts of the Mediastinum

A dermoid or a hydatid cyst is occasionally met with (see also p 192) Treatment is surgical Mesothelial mediastinal cysts are not very uncommon They are also known as spring water cysts from the clear fluid they contain or pericardial celomic cysts They probably arise from a fold in the advancing pleural cavity at the 15 mm embryo stage The fold is cut off and connective tissue laid down around it They usually occur in the anterior inferior mediastinum and radiologically are seen as a rounded shadow in the cardio phrenic angle on the right side They are easily removed surgically The differential

diagnosis includes the following a fat pad in the cardio phreni sulcus, a lipoma, a dermoid a teratoma a neurofibroma a diaphra matic hernia, a hump of the antero mesial portion of the diaphragm cardiac aneurysm unilateral sacculated pericarditis a hamartoma a primary or secondary carcinoma of the lung

## THE DIAPHRAGM

### Hiccough

### (*Singultus*)

**Etiology** Sudden diaphragmatic contractions with reflex closure of the glottis may be due to alimentary nervous or renal causes 1 Alimentary causes include *Irritation of the œsophagus or stomach* as by tobacco smoke and pungent articles of food, hiccough may also occur in association with dilatation of the stomach gastritis, enteritis peritonitis or intestinal obstruction 2 Nervous causes may be central such as cerebral tumour meningitis epilepsy encephalitis lethargica or hysteria or peripheral such as a reflex from a mediastinal tumour pericardial effusion or diaphragmatic pleurisy 3 Renal causes as in chronic nephritis or uræmia *Tonic spasm* of the diaphragm may be due to strychnine poisoning tetanus hydrophobia or laryngismus stridulus

**Treatment** If possible the cause should be removed The clonic spasm may sometimes be abolished by holding the breath or by *drinking a glass of water* In severe cases it may be necessary to prescribe sedatives such as phenobarbitone gr ½ to 1 t d s or the hypodermic injection of diamorphin hydrochlor gr ⅛ Inhalation of the vapour from a capsule of amyl nitrite may prove successful $CO_2$ may be inhaled by breathing in and out of a paper bag held tightly over the nose and mouth or 5% $CO_2$ and oxygen may be inhaled through a mask for 5 or 10 minutes The intravenous injection of mg 50 of chlorpromazine may cause an immediate cessation of hiccough The *phrenic nerve* may be injected with procaine or the effect of spraying the skin of the neck over the phrenic nerve with ethyl chloride may be tried, if the condition is unilateral as shown by X ray examination

### Diaphragmatic Pleurisy

This has been referred to on p 194

### Diaphragmatic Paralysis

**Etiology** This may be due to central causes affecting the phrenic nuclei such as poliomyelitis a hæmorrhage or a tumour The phrenic nerve may be involved by neuritis or by pressure of a tumour or it may be crushed or divided surgically or after division avulsed from its diaphragmatic terminations

**Clinical Findings** X ray examination will show the extent of diaphragmatic movement One half may be completely paralysed in which case the diaphragm on that side is usually raised or it may show paradoxical movement on the affected side, rising with inspiration and

falling with expiration This is often associated with a mediastinal neoplasm

### Hernia of the Diaphragm

*(Thoracic Stomach)*

**Definition** Protrusion of an abdominal viscus usually the stomach through the diaphragm into the thoracic cavity

**Etiology** Diaphragmatic hernia may be traumatic or non traumatic Sixteen per cent are traumatic The non traumatic ones are either congenital or acquired The congenital ones are usually due to absence of a part of the diaphragm and are met with in infants The non traumatic ones are due usually to developmental diaphragmatic defects and show themselves later in life They are subdivided into 1 Œsophageal hiatus hernia This may be a true hernia with a sac or a false hernia due to a congenital short œsophagus 2 Pleuro peritoneal hernia the weakness of the diaphragm occurring at the site of the foramen of Bochdalek 3 Postero lateral diaphragmatic hernia 4 Anterior herniation through the site of the foramen of Morgagni

**Clinical Findings** The symptoms in infancy are cyanosis dyspnœa and vomiting In adults the symptoms may be mistaken for those of peptic ulcer cholecystitis œsophageal or intestinal obstruction anæmia or coronary occlusion Hæmatemesis may occur or the occult blood test on the fæces be positive The diagnosis is established by a barium swallow and meal with the patient in the Trendelenburg position

**Treatment** Immediate surgical repair is required for diaphragmatic hernia in infants In adults small symptomless herniæ require no treatment With moderate symptoms the diet should be bland with small and frequent meals Neutralising substances are required for heartburn Phrenic crush is often very successful in quickly checking the bleeding causing anæmia and in relieving the coronary type of pain For more severe types of hernia surgical repair is necessary

### Eventration of the Diaphragm

**Definition** A bulging of the affected side of the diaphragm into the thorax

**Etiology** The eventration is probably due to a congenital weakness of the diaphragm rather than to an affection of the phrenic nerve but in some cases there may be a birth injury of the phrenic nerve Acquired cases result from lesions of the phrenic nerve often due to poliomyelitis or after pneumonectomy

**Pathology** The affected half of the diaphragm is thin translucent and consists of fibrous tissue with a few muscle fibres The phrenic nerve on the affected side may be smaller than its fellow but does not show evidence of neuritis In about 94% of cases the left side of the diaphragm is affected

**Clinical Findings** The patient does not usually make any complaint pointing to a lesion of the diaphragm, but the signs found on examination may resemble those of a diaphragmatic hernia The lesion is diagnosed by X ray examination, the affected side of the diaphragm may lie at the level of the third costal cartilage, and show paradoxical movement

**Treatment** No special treatment is required

### Paroxysmal Flutter of the Diaphragm

This is a rare condition in which the symptoms resemble those of angina pectoris Screen examination shows rapid oscillations of one half of the diaphragm, superimposed on somewhat jerky movements synchronous with thoracic respiration Rapid relief has been recorded by spraying the skin of the neck over the phrenic nerve with ethyl chloride

## CHAPTER III

## THE CARDIO-VASCULAR SYSTEM

**Introductory** In this section attention will be directed to the pericardium heart arteries and veins The indications for special investigations will be noted These include X ray examinations to determine the size shape position and movements of the heart and aorta electrocardiograms cardiac catheterisation and angiocardiography

### The X-ray Appearances of the Heart

The size and outline of the heart shadow can be determined either by a teleradiogram or by an orthodiagram In the former an X ray photograph is taken with the tube at a distance of 2 metres (6 feet) from the patient so that there is little distortion of the heart shadow With the orthodiagram a small beam of rays is moved round the outline of the heart and its edge can thus be delineated with a pencil on the

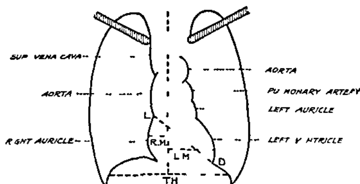



Fig 12 Diagram of Normal Orthodiagram—P-A View

*Average Measurements* L.D = Long diameter = 13 2 cm R.M = Rt medial = 4 2 cm L.M = Lt. medial = 8 5 cm T D = Transverse diameter = R M + L M TH. = Internal diameter of thorax. Cardio thoracic ratio $= \frac{TD}{TH} = \frac{1}{2}$

screen or on paper The outline is drawn in expiration and during the diastolic phase of cardiac activity A diagram showing the main features of a normal heart shadow is given (see Fig 12) X ray examinations in the postero anterior and oblique positions will show alterations in the size and shape of the heart of its chambers and of the aorta It will also reveal calcification of the heart aorta or pericardium and erosion of ribs by the hypertrophied intercostal arteries in some cases of coarctation of the aorta

X-ray Kymography This provides a record on a film of the movements of the heart and aorta A grid made of a lead plate with narrow horizontal slits equidistant from each other is placed between the patient and the film The grid moves slowly downwards during the exposure which lasts 8 seconds The heart outline appears on the film with a serrated edge, each serration corresponding with a beat of the heart Diminution or absence of pulsations of a part of the heart as may occur with a cardiac aneurysm, results in diminution or absence of serrations over the corresponding portion of the heart outline Kymography appears of value chiefly in the diagnosis of a cardiac aneurysm or pericardial effusion or in differentiating a pulsating mediastinal tumour from an aortic aneurysm

*The Normal Electrocardiogram* This is illustrated in Fig 13 The P wave is due to auricular systole and constitutes the auricular

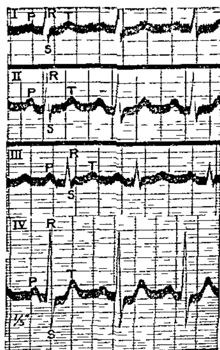



Fig 13 Normal Electrocardiogram (Lead IVR left arm electrode anteriorly right arm electrode on right arm)

complex QRST occur during ventricular systole and constitute the ventricular complex Q and S are often absent T to P represents diastole

Angiocardiography 45 to 60 ml of 70% diodone are injected into the basilic vein in adults and about 20 to 30 ml into the saphenous vein in children Eight to ten X ray exposures of the chest are made over about 8 seconds beginning at the commencement of the injection Postero anterior and occasionally oblique views are taken

## THE PERICARDIUM

### Acute Fibrinous Pericarditis

(*Dry Pericarditis Plastic Pericarditis*)

**Definition** Acute inflammation of the pericardium with little or no fluid formation

**Etiology** *Varieties* The chief varieties of pericarditis are rheumatic pyogenic tuberculous, traumatic secondary to myocardial infarction uræmic and associated with adjacent malignant lesions A benign type is also described complicating upper respiratory infections which occurs chiefly in young men It is sometimes met with in scarlet fever meningococcal meningitis actinomycosis undulant fever primary atypical pneumonia disseminated lupus erythematosus polyarteritis nodosa and myxœdema The exciting organisms include the Diplococcus pneumoniæ (pneumococcus) the streptococcus the staphylococcus the Neisseria gonorrhœa (gonococcus) the Bacterium commune (Bact coli) the Bacterium typhosum (B typhosus) and the Mycobacterium tuberculosis (B tuberculosis)

**Pathology** A fibrinous exudate forms which is localised or diffuse There may be a shaggy layer giving a bread and butter effect A small serous exudate is not uncommon Evidence of an old healed lesion is sometimes found at autopsy in the form of a thickened white area or ' milk spot '

**Clinical Findings** If the patient is a child he is frequently suffering from rheumatism or chorea dry pericarditis in an adult may be due to any of the causes mentioned above whereas in old age it is often a terminal event Pain is complained of if the inflammation affects the lower third of the pericardium which is supplied by the phrenic nerve or if the pleura is also involved It is localised to the præcordium but at times radiates to the neck arm or abdomen It is generally intensified by breathing coughing or moving In some cases only slight discomfort or a dull ache is noticed

*On Examination* The patient usually looks ill and anxious and he may be pale The temperature is raised to about 101 F and the pulse rate to 100 or 120 The chest Inspection A rapidly beating cardiac impulse is often seen Palpation A to and fro pericardial friction rub may be felt Percussion The cardiac dulness is sometimes but not always enlarged but such enlargement is not due to the pericarditis Auscultation The diagnostic friction rub is heard This is a to and fro ' rub not quite synchronous with systole and diastole and usually intensified by pressure of the stethoscope It may be very faint or loud and creaking (*bruit de cuir neuf*) and audible without a stethoscope It may be diffuse or localised to the base or apex of the heart In some cases it is heard better on deep inspiration or when the patient is sitting Further it is sometimes inconstant being heard at one examination and not at the next The electrocardiogram usually shows an early elevation of the S T segment which resumes the iso potential level in a few days and is followed by inversion of the T wave especially in lead II

**Differential Diagnosis** The pain must be distinguished from that due to pleurisy or to angina pectoris Dry pericarditis is often a complication of coronary obstruction The rub has to be differentiated from pleuro-pericardial or pleural friction, and from a double aortic murmur With pleuro pericardial friction the rub is generally heard at the left or right border of the heart disappearing with full inspiration and returning with expiration With pleural friction the rub may disappear completely when the breath is held With a double aortic murmur the characteristic features are the site and area of conduction of the murmur, the absence of pain, the blood pressure readings and the cardiac enlargement

**Course and Complications** In the majority of cases the pericarditis resolves without the formation of an effusion which can be detected clinically In some instances a serous, hæmorrhagic or purulent pericardial exudate develops Adherent pericardium is a likely sequela

**Prognosis** This must vary with the cause but pericarditis is always a serious condition and if death does not ensue time alone will show what permanent damage will remain In chronic nephritis pericarditis is usually a signal of impending death

**Treatment** The patient must be kept strictly at rest in bed lying nearly flat or slightly propped up or turned a little to one side or the other Ung methyl salicyl (B P C ) should be applied on lint daily to the præcordium in rheumatic cases Pain is relieved by the injection of morphin sulph gr 1/6 to 1/4 or of pethidine hydrochlor mg 100, or by pulv ipecac et opii gr 10 taken by mouth for an adult at night The treatment is that of the underlying cause In rheumatic cases a mixture containing Sod salicyl gr 10 sod bicarb gr 20 syr aurant, m 30 aquam ad fl oz 1 is given fl oz 1 t d s p c A careful watch should be kept as signs of collapse may occur Should this happen the salicylate mixture is discontinued and cardiac stimulants should be given such as Coramine 1 5 ml subcutaneously every 4 hours The diet should be fluid or semi solid during the acute stage The patient must be kept in bed for at least 3 months after the temperature and pulse have returned to normal without salicylates being taken Subsequently very limited walking exercise is allowed for 9 months and no games should be played for 2 years

### Pericardial Effusion

The following varieties may occur 1 Clear Sero fibrinous pericarditis Hydropericardium 2 Purulent Pyopericardium 3 Hæmorrhagic Hæmopericardium Hæmoseropericardium Hæmohydropericardium

### Pericarditis with Effusion

*(Sero fibrinous Pericarditis Seropericardium)*

**Definition.** An inflammatory serous pericardial exudate

**Etiology** Pericarditis with effusion is usually preceded by acute fibrinous pericarditis but in some cases, especially in children it develops insidiously

**Pathology** The fluid varies in amount from a few ounces to 2 or 3 pints It is yellowish green in colour clear or slightly turbid, specific gravity about 1 018 and it clots on standing The pericardium is thickened by a fibrinous exudate and there is usually myocardial degeneration

**Clinical Findings** The patient is a child or an adult Usually there is a history of acute dry pericarditis in the course of which the effusion occurred Rarely the pericardial effusion is discovered without such a history the patient being a child who is off colour looks pale and complains of shortness of breath The patient with such an effusion complains chiefly of dyspnœa a feeling of distress or actual pain in the præcordium and palpitations Increasing mediastinal pressure may result in dysphagia vomiting hiccough cough or hoarseness due to recurrent laryngeal paralysis Insomnia is often a very troublesome feature

*On Examination* The patient is pale and the lips and ears may be somewhat cyanosed He is often restless and cannot lie flat on his back wishing to be propped up or turned on the left side The cervical veins may be distended The chest Inspection There may be bulging of the præcordium with diminished expansion on the left side of the chest Often no cardiac impulse is visible but a diffuse wave may be seen over the third fourth and fifth left intercostal spaces Diaphragmatic movement may be abolished or weakened on the left side Palpation The cardiac impulse is feeble or absent Pericardial friction if previously present disappears Œdema of the chest wall over the præcordium may be demonstrated by pitting on pressure Percussion The area and intensity of the cardiac dulness are increased It is described as pear shaped with the stalk upwards The upper level of dulness may alter with the patient in the erect and recumbent positions The dulness obliterates the normal area of pulmonary resonance in the cardio phrenic angle at the fifth right space near the sternum (Rotch s sign) On the left side the dulness may extend out to the axilla Auscultation The heart sounds become progressively weaker as the effusion increases A friction rub if previously present disappears although it may remain at the base or be audible in the erect but not in the recumbent position The apex of the heart as judged by the intensity of the heart sounds lies internal to and usually above the apex of the cardiac dulness The lungs The pressure of the pericardial effusion especially if localised to the posterior part of the pericardial sac may give rise to special signs Thus a small area of dulness may be found near the inferior angle of the left scapula with bronchial breathing bronchophony and whispering pectoriloquy (Bamberger s or Ewart s sign) The pulse is usually rapid and it may increase in force with expiration and weaken with inspiration (the pulsus paradoxus) Cardiac tamponade is the term applied to circulatory failure due to rise of intrapericardial pressure The venous pressure rises the blood pressure falls the pulse rate rises and there is peripheral vasoconstriction The blood usually shows a leucocytosis An X ray examination will generally reveal a characteristic shadow of the effusion This is globular

in the recumbent position and pyriform when the patient is erect With a dilated heart the shadow remains practically unaltered in shape with change of posture The absence of normal pulsations is shown by X ray kymography

**Differential Diagnosis** A pericardial effusion may be mistaken for a dilated heart or for a left sided pleural effusion In dilatation of the heart the apex beat is usually palpable although weak The dulness over the heart is not so stony in character and Rotch's sign is not present The shape of the dulness is also different the upper border being more flat and at a lower level The heart sounds are more distinctly heard pressure signs are not found behind at the left base and the X ray shadow differs as described above If a cardiac catheter is inserted into the right auricle X ray examination will show that its tip does not extend to the edge of the shadow cast by the heart and pericardial effusion With a left sided pleural effusion the heart is displaced towards the right the heart sounds are clear and unless the fluid is loculated, the signs of fluid are found at the left base behind X ray examination will also serve to differentiate

**Course and Complications** With recovery the fluid absorbs and adhesions remain between the layers of the pericardium The rate of absorption varies from a few days to several weeks Reappearance of the friction rub is an indication that this has taken place and pain may or may not recur In some instances the fluid becomes purulent

**Prognosis** A pericardial effusion is always a serious event the immediate prognosis varying with the underlying cause Thus with the infectious fevers recovery usually occurs but in severe septic infections a pericardial effusion is generally the herald of death

**Treatment** The general treatment is as for acute fibrinous pericarditis Special treatment involves paracentesis of the pericardium This should not be lightly undertaken as the results are very discouraging It may be necessary if the effusion increases and seriously embarrasses the heart s action as shown by increasing rapidity and weakness of the pulse greater dyspnœa and restlessness or if pus is suspected Procaine 1 ml of a 2% solution should be injected in the fourth left space close to the edge of the sternum or 1 inch external to it in order to avoid the internal mammary artery until the pericardium is pierced when the fluid can be withdrawn into the syringe A fine trocar and cannula are then inserted and the fluid allowed to drain away If however pericardial friction is audible at this site the puncture should be made in the angle between the xiphoid cartilage and the left costal cartilages the needle then being passed upwards and backwards at an angle of 30 to the skin until the pericardium is pierced Alternatively the needle may be inserted in the fifth or sixth left space just medial to the left border of the pericardial dulness A loculated posterior pericardial effusion may be aspirated from the back The left arm is brought forward and the needle is inserted in the seventh or eighth left space in the mid scapular line to a depth of 2 or 3 inches

### Hydropericardium

**Definition** A transudate of fluid into the pericardium

**Etiology** Hydropericardium is most frequently associated with heart failure or chronic nephritis Rarely it is due to mediastinal venous obstruction from enlarged glands new growths or aneurysm or to the severe cachexia of leukæmia or of beri beri

**Pathology** The fluid is clear pale of low specific gravity (about 1 012) and contains less than 3% of protein

**Clinical Findings** The symptoms are usually masked by those of the primary condition A considerable amount of fluid may accumulate without definite symptoms as there is no preliminary stage of pericardial friction and the rate of output of the transudate is slow The physical signs correspond with those described for sero fibrinous pericarditis There is usually œdema in other parts of the body

**Treatment** It is rarely necessary to tap the pericardium and the treatment is that appropriate to the causative condition

### Purulent Pericarditis

(*Pyopericardium*)

**Definition** Pus in the pericardium

**Etiology** Purulent pericarditis is usually a complication of pyæmia, especially when there is osteomyelitis or it is associated with tuberculosis or new growths of the pericardium An empyema or pulmonary abscess may lead to direct spread of infection to the pericardium *Tuberculous pericarditis is usually secondary to medi astinal gland tuberculosis*

**Clinical Findings** In addition to the symptoms and signs of sero fibrinous pericarditis the temperature shows marked diurnal variations the pulse is more rapid and sweats or rigors may occur There is generally a leucocytosis of over 12 000 per c mm In rare cases the patient is afebrile

**Differential Diagnosis.** The nature of the fluid can only be determined by exploratory puncture

**Course and Complications** The disease is usually rapidly fatal but the course may be very prolonged in tuberculous infections and there may be calcification of the pericardium The fluid may rupture through to the left pleura with empyema formation

**Prognosis** This is very grave Some ca es of tuberculous pyoperi cardium undergo a spontaneous cure

**Treatment** The pericardial sac should be aspirated the needle being inserted between the xiphoid cartilage and the left costal cartilages in order to avoid infecting the pleura A course of penicillin should now be given 600 000 units of a procaine preparation every 12 hours intramuscularly for 3 to 4 weeks If the patient s general condition permits and the fluid is not tuberculous the pericardium should be drained surgically

### Hæmopericardium

**Definition** Blood in the pericardium

**Etiology** The blood enters the pericardium from rupture of the heart, coronary artery or intrapericardial portion of the aorta The cardiac leak may result from an infarct an aneurysm of the heart or it may be due to external trauma or to a perforating œsophageal wound Hæmopericardium is a rare occurrence in purpura or scurvy

**Clinical Findings** The patient suddenly dies

### Hæmoseropericardium

***Definition*** *An inflammatory serous pericardial exudate containing blood*

**Etiology** Hæmoseropericardium is usually due to malignant disease or tuberculosis of the pericardium

**Clinical Findings** These closely resemble the findings in sero fibrinous pericarditis

### Hæmohydropericardium

**Definition** A pericardial transudate, containing blood

**Etiology** Hæmohydropericardium is usually associated with heart failure

### *Calcified Pericardium*

This may be a sequela of acute inflammatory pericarditis especially pyopericardium, or of a chronic pericardial effusion It is usually discovered by X ray examination It may cause no symptoms or result in cardiac distress

### *Pyopneumopericardium*

**Definition** Gas and pus in the pericardium

**Etiology** The gas may come from various sources such as 1 The exterior In trauma from wounds or by operation on the pericardium 2 The lungs In tuberculosis carcinoma, gangrene or pyopneumothorax 3 The abdomen In subphrenic abscess 4 The œsophagus With an ulcerating growth 5 Gas producing organisms in a pericardial effusion This is generally a post mortem event

*Pathology* *A purulent exudate is present*

**Clinical Findings** The patient may complain of sudden præcordial pain but at times the onset is insidious

*On Examination* Typical signs depend upon the presence of gas and fluid in the pericardium There is an area of hyperresonance over the præcordium which may move with alteration in the position of the patient On auscultation a loud churning sound is heard

**Differential Diagnosis** If gas alone is present it may be loculated behind the heart and cause no definite signs When in front of the heart the *hyperresonance may suggest* acute interstitial emphysema which has spread to the anterior mediastinum An X ray examination will establish the diagnosis the outline of the pericardium standing out as a definite line with a translucent space between it and the heart shadow

**Treatment** This is similar to that described for acute sero fibrinous pericarditis The air may be allowed to escape through a needle if there is severe cardiac distress

### Pneumopericardium

This may complicate artificial pneumothorax treatment, and is probably due to a congenital pleuro pericardial communication

### Adherent Pericardium

**Definition** Adhesions between the layers of the pericardium, or between the pericardium and surrounding structures

**Etiology** Non-constrictive adherent pericardium is usually a sequel of acute pericarditis The majority of cases are therefore rheumatic the minority being caused by tuberculosis sepsis cardiac infarction trauma malignant disease and polyserositis (Concato s disease)

**Pathology** There are four varieties 1 Adhesions are present between the visceral and parietal pericardial layers 2 The adhesions also extend loosely outwards to the diaphragm chest wall and pleura (pericarditis interna et externa) 3 The pericardium becomes solid and thick or even calcified 4 There is also thickening of the media stinal connective tissue (chronic indurative mediastino pericarditis) The last two varieties are called constrictive pericarditis They impede the cardiac output and obstruct the venous inflow The heart is not grossly enlarged unless valvular disease or hypertension is present

**Clinical Findings** Unless there is constrictive pericarditis the adhesions usually give rise to no symptoms Broadbent s sign indicates extra pericardial adhesions to the chest wall and diaphragm Systolic retraction is seen in the tenth or eleventh space behind on the left side in the scapular angle line No special treatment is required unless constrictive pericarditis is present

### Chronic Constrictive Pericarditis

*(Pick s Disease)*

**Etiology** Rheumatism is not a cause The majority of cases are due to tuberculosis a few are due to sepsis and in others the cause is not known

**Pathology** Pick in Germany described pericarditic pseudocirrhosis of the liver in 1896 A similar condition was described by Chevers at Guy s Hospital in 1842 There is pericarditis enlargement of the liver perihepatitis and ascites

**Clinical Findings** The patient complains of shortness of breath swelling of the abdomen and of the legs

*On Examination* The upper half of the body is often thin but the abdomen is very swollen The cervical venous pressure is raised the liver is enlarged there is ascites and œdema of the legs The pulse is small and may weaken during inspiration and become stronger with expiration (pulsus paradoxus) There is peripheral vaso constriction with pallor cyanosis and coldness of the extremities The blood pressure

is usually low The heart is often not enlarged or only moderately so, and X ray examination of the heart shows diminished pulsation Calcification of the pericardium is seen in some cases The electro cardiogram shows low voltage curves with flat or slightly inverted T waves

**Treatment** Pericardial resection is indicated to relieve the com pression of the heart and great veins Before operation a pleural effusion should be aspirated ascites tapped and mersalyl given The operation, however, should not be deferred until there is no ascitic fluid In carefully selected cases a *cure may be expected in about 50%* but the operative mortality is about 10%

### New Growths and Cysts of the Pericardium

The pericardium may be invaded by an endothelioma of the pleura by spread of carcinoma or by a primary sarcoma A hydatid cyst sometimes develops in the pericardium and a dermoid cyst of the mediastinum may rupture into it

## THE NEURO MYOCARDIUM

### Disorders of Rate and Rhythm

**Introductory** Normally the impulses for the heart beat arise in the sino auricular node in the right auricle whence waves of excitation *spread over both auricles to the auriculo ventricular node* situated at the posterior and right edge of the interauricular septum The impulse then spreads to the ventricles along the bundle of His The S A node is under a double nervous control the sympathetic accelerating and the vagus retarding the output of stimuli Normal cardiac activity *is* therefore a sinus rhythm Cardiac irregularities may be due to 1 Sinus disorders Simple tachycardia Simple bradycardia and sinus arrhythmia 2 Increased muscular irritability Paroxysmal tachy cardia Premature systoles Auricular fibrillation Auricular flutter Ventricular fibrillation 3 Diminished conductivity Heart block 4 Defective contractibility Pulsus alternans 5 Disturbed diastolic filling Pulsus paradoxus These varieties will now be briefly considered

### Simple Tachycardia

**Etiology** Simple tachycardia may result from diverse causes such as exercise emotion fevers hyperthyroidism chronic infections anæmia hæmorrhage alcohol cordite atropine tea coffee tobacco and thyroid extract It may be due to a rise of pressure in the right auricle causing a release of vagal tone or to stimulation of the pacemaker in the heart

*Clinical Findings* The pulse rate at rest is usually between 90 and 120 and after exercise it may rise to 150 or 180 taking longer than 2 minutes to return to its resting rate In such a case the exercise tolerance is considered fair, moderate or poor, according to the pulse figures and the *distress engendered by the test* In other instances there are attacks of palpitations during which the patient is conscious of the

heart s action Throbbing may be felt in the neck and there may be giddiness or buzzing in the ears A sense of præcordial distress or pain may also be noticed

*On Examination* There is usually no cardiac enlargement A soft systolic murmur heard at the apex or base may accompany the first sound

**Treatment** Digitalis is not usually of value unless an organic cardiac lesion is present Bromide in the form of ammon brom gr 5 to 10 t d s is often helpful A search should always be made for an underlying cause such as a septic focus pulmonary tuberculosis or hyperthyroidism

## The Effort Syndrome

*(Da Costa s Syndrome Cardiac Neurosis Disorderly Action of the Heart D A H Soldier s Heart Neuro-circulatory Asthenia)*

**Etiology** This syndrome is a frequent occurrence in wartime amongst untrainable or imperfectly trained soldiers In civilian life it is said to be more common in women and it also occurs in children Various views are held as to its nature 1 That it is a neurosis 2 That it is due to chronic sepsis 3 That it results from over smoking 4 That it is the product of effort and poor physique It is unlikely that effort alone will cause it and the symptoms do not resemble those produced by strenous exercise in a healthy person In the majority of cases there is a mental background of fear anxiety or a compensation neurosis and the physical condition is often poor

**Clinical Findings** The patient is usually a young adult who complains of breathlessness sighing palpitations fatigue sweating nervousness dizziness and left inframammary pain

*On Examination* No signs of organic disease are found The physique is often poor there is vasomotor spasm of the extremities as evidenced by cold or bluish red hands and feet and abnormal sweating is seen in the axillæ and on the hands and feet The heart The apex beat may be forcible but there is usually no cardiac enlargement The heart sounds are normal but the first sound may be accentuated if there is tachycardia A short soft systolic murmur may be heard at the apex or base The average resting pulse rate is between 80 and 100 The blood pressure is usually normal The exercise tolerance may be impaired but is often normal

**Differential Diagnosis** Such conditions as rheumatic carditis early mitral stenosis thyrotoxicosis pulmonary tuberculosis pleurisy angina pectoris hypoglycæmia and malingering must be excluded A carefully taken history will indicate the correct diagnosis in the majority of cases and this is confirmed by the characteristic symptoms and signs

**Prognosis** This is unfavourable only about 25% of soldiers suffering from the syndrome can be rendered fit for full military duties Medical boards should not enlist neurotics who will never make efficient soldiers

**Treatment** When soldiers are so enlisted however an attempt should be made to improve their condition Properly graduated

exercises should be first given to increase the cardio respiratory efficiency The patient should be informed of the nature of his complaint and treated as a psychoneurotic

## Simple Bradycardia

**Etiology** Simple bradycardia may be due to vagotonia occurring during convalescence from severe illnesses such as influenza or typhoid fever It is also met with in association with starvation nervous exhaustion cerebral abscess tumour or hæmorrhage, meningitis, myxœdema jaundice uræmia vaso vagal attacks overdosage of digitalis and at times with myocardial degeneration Irritation of the vagus by a mediastinal tumour may also cause bradycardia Physiological bradycardia is met with in tall athletes, in adolescence and old age

**Clinical Findings** The apex rate varies between 40 and 60 The electrocardiogram shows no evidence of heart block

**Treatment** No special treatment is required beyond that indicated for the underlying condition

## Sinus Arrhythmia

*(Physiological or Juvenile Cardiac Irregularity)*

**Etiology** Sinus arrhythmia is met with in young people and during convalescence from febrile illnesses It is due to alteration of vagal tone with respiration and indicates that the impulse for the heart beat arises in the sino auricular node

**Clinical Findings** The patient is usually a young adult Sinus arrhythmia generally causes no symptoms but when discovered it may be mistaken for some serious cardiac irregularity The pulse rate is found to slow during expiration and quicken during inspiration This may occur periodically and only be detected when the patient is asked to breathe slowly and deeply The irregularity disappears when the heart beats rapidly as after exercise The electrocardiogram shows that the alteration is due to variations in the length of diastole

**Treatment** Sinus arrhythmia in itself is of no pathological import and requires no treatment but it may be associated with all varieties of heart disease

## Paroxysmal Tachycardia

The following varieties are described 1 Simple paroxysmal tachycardia 2 Nodal tachycardia 3 Ventricular tachycardia 4 Paroxysms of auricular flutter 5 Paroxysms of auricular fibrillation

### Simple Paroxysmal Tachycardia

**Etiology** The paroxysms of tachycardia are probably due to impulses arising at a new focus in the auricle, constituting a regular series of premature systoles Some cases are associated with pregnancy, others remit during pregnancy Exertion emotion or flatulence may induce an attack

**Clinical Findings** The patient complains of periodical attacks of palpitations which may be accompanied by faintness dyspnœa and præcordial distress

*On Examination* The patient may be pale somewhat cyanosed and sweating The apex and pulse rate vary usually between 140 and 250 The rhythm is regular and the rate is not slowed on lying down The systolic blood pressure falls and the output from the heart is diminished In severe cases signs of cardiac decompensation are evident such as dilatation of the heart œdema of the lungs and legs and engorgement of the liver The electrocardiogram shows a rapid regular rhythm with normal ventricular complexes The P wave is often inverted in leads II and III and modified in lead I

**Differential Diagnosis** The sudden onset and cessation and the constancy of the rate with exercise rest and change of posture differentiate it from simple tachycardia The engorgement of the liver may suggest an abdominal lesion, or the signs at the bases of the lungs may simulate pneumonia The history signs and electrocardiogram establish the diagnosis

**Course and Complications** The attack lasts from a few minutes to two weeks and then stops abruptly Blindness and epileptiform convulsions may occur temporarily Death during an attack has been recorded in a few cases

**Treatment** The following devices may be employed to abort the attack Holding the breath stooping down and breathing deeply pressure over one carotid sinus but not over both at the same time pressure on the eyeballs a tight abdominal binder ice applied to the præcordium swallowing a hard article of food such as a crust of bread or the induction of vomiting by faucial irritation Cholinergic drugs such as methacholin chlorid (Mecholyl chloride) mg 10 to 25 may be injected subcutaneously or a carbachol tab *mg* 2 taken by mouth If vomiting defæcation or faintness is produced by the drug atropin sulph gr 1/100 should be injected intravenously If the paroxysm persists quinidine should be given as for auricular fibrillation Later quinidine gr 3 may be taken daily to prevent attacks

### Nodal Tachycardia

Paroxysms of premature systoles arise in the A–V node resulting often in a simultaneous contraction of the auricles and ventricles In the electrocardiogram the P–R interval may be shortened and the P wave inverted or the P and R waves are fused or the P wave may follow the R wave The clinical picture and treatment resemble those of simple paroxysmal tachycardia

### Ventricular Tachycardia

This is a rare condition due to a regular sequence of premature ventricular systoles The ventricles may contract 180 times a minute while the auricular rate remains at about 80 It is usually associated with severe myocardial damage coronary occlusion or gross digitalis poisoning The clinical features closely resemble those of the other

varieties of simple paroxysmal tachycardia but the ventricular beats are often slightly irregular Treatment consists in giving quinidine gr 5 by mouth every 2 hours up to a total of gr 40 in a day Alternatively Pronestyl (procaine amide hydrochloride) can be given by mouth, if the patient can swallow, the dose being 4 capsules (0 25 G each) followed by 2 capsules every 4 to 6 hours until the paroxysm stops For very urgent cases an intravenous injection can be given slowly, not more than 1 ml (mg 100) per minute up to a total of 10 ml The blood pressure should be taken in the other arm during the injection and, if it falls unduly the injection should be stopped Pronestyl is probably successful in restoring a normal rhythm in over 50% of cases In some cases it may lead to leucopenia Digitalis should not be given

### Paroxysms of Auricular Flutter

This is a common cause of paroxysmal tachycardia The apex and pulse rate are about 160, the rate is uninfluenced by posture and the rhythm is usually regular The auricular rate is about 200 to 300 Diagnosis is established by means of the electrocardiogram The treatment is as for auricular flutter

### Paroxysms of Auricular Fibrillation

The apex and pulse show the typical irregularity of auricular fibrillation and the electrocardiogram confirms the diagnosis The treatment consists in the administration of quinidine as for auricular fibrillation

### Premature Systoles

*(Ectopic Beats)*

**Definition** Interpolation on the normal sinus rhythm of premature contractions arising at some other focus in the heart than the sino auricular node

**Etiology** Premature systoles are believed to be due to undue excitability of the auricle A–V node or ventricle They are commonly met with in middle age associated with myocardial degeneration In young people they may occur without any evidence of cardio vascular lesions They may be associated with oversmoking neurasthenia pregnancy the administration of digitalis or aconite or coal gas poisoning There are three main varieties auricular nodal and ventricular In about 70% of cases they are ventricular in origin

**Clinical Findings** The patient may be quite unaware of any cardiac irregularity or he may be conscious of a pause in the cardiac rhythm as if the heart had missed a beat This is particularly noticeable when the patient is in bed He may also complain of palpitations

*On Examination* The pulse may either show a pause due to the premature beat being of insufficient force to reach the wrist (intermittent pulse) or a weak beat may be felt On listening over the apex a premature and weak contraction will be heard at the moment that the beat is missing or weak at the wrist An occasional premature

contraction may occur or they may be frequent causing a gross irregularity *Pulsus bigeminus* which may be met with in overdosage of digitalis consists of coupled beats due to premature systoles The electrocardiogram will show typical curves and indicate the site of the irregularity

**Differential Diagnosis** Premature systoles must be diagnosed from heart block or a slow auricular fibrillation In heart block when the pause is felt at the wrist, there is silence on auscultation over the apex Premature systoles are usually diminished in frequency or abolished by exercise but with a slow fibrillation it may be impossible to establish the diagnosis without an electrocardiogram

**Prognosis** Premature systoles can usually be disregarded in young people Later in life, especially if there is evidence of cardio-vascular degeneration they are generally indicative of myocardial degeneration

**Treatment** Young people should be assured that there is no cause for worry The patient should not smoke Quinidine gr 3 t d s will often ensure a regular rhythm If there is real distress particularly at night ammon brom in doses of gr 5 to 10 should be given t i d

## Auricular Fibrillation

**Definition** A condition characterised by flickering auricular contractions of a peculiar type with an irregular ventricular response

**Etiology** Auricular fibrillation may be associated with infections *especially with rheumatism and less frequently with scarlet fever* diphtheria influenza and pneumonia Mitral stenosis is often associated with fibrillation and it may occur in association with an auricular septal defect Later in life it may be met with in ischæmic heart disease and in tumours of the heart or lungs It may occur also in Graves disease and in hypertensive heart disease

**Pathology** Prinzmetal says there are two types of auricular con traction seen in experimental auricular fibrillation (*a*) minute irregular and localised contractions and (*b*) large rhythmic contractions passing across the auricle at a rate of 400 to 600 a minute

**Clinical Findings** The disturbance produced varies with the frequency of ventricular contractions If there is a slow fibrillation there may be no subjective symptoms but when the ventricle contracts rapidly cardiac decompensation usually ensues The patient may complain of palpitations or of præcordial discomfort dyspnœa and swelling of the extremities

*On Examination* The patient may or may not be dyspnœic at rest or on slight exertion according to the degree of heart failure present The presystolic murmur of mitral stenosis disappears with the onset of fibrillation if the heart is beating slowly The rhythm is character istically completely irregular the beats also varying in intensity so that many of the ventricular contractions fail to cause a pulse at the wrist The pulse This is completely irregular and its rate is usually slower than that of the ventricle Records of pulse rates are therefore quite valueless in determining the rate of the heart The lungs liver abdomen

*and extremities may show* signs of venous stasis and œdema The electrocardiogram is typical, the P waves are absent and replaced by F waves, and the ventricular complexes are irregular in rate and degree

**Differential Diagnosis** Auricular fibrillation can usually be readily diagnosed by examination of the heart and pulse *Without a tracing* slow fibrillation is difficult to differentiate from premature systoles

**Course and Complications** Auricular fibrillation may occur in paroxysms the normal rhythm being restored spontaneously from time to time In other cases, and despite treatment it persists for indefinite periods, or the normal rhythm may be quickly restored by adequate treatment Complications include congestive heart failure and systemic embolus formation The latter is especially liable to follow restoration to normal rhythm by the administration of quinidine, a portion of clot being detached from the left auricle The main arterial supply to a limb may be obstructed with severe pain The limb *becomes cold and blue and gangrene may supervene*

**Prognosis** This always grave although a patient may live for over 10 years with auricular fibrillation

**Treatment.** Digitalis is usually prescribed as tablets of the powdered leaf or as digoxin For a patient who is not seriously ill tab digital præp gr 3 may be given t i d on the first day gr 2 t i d on the second day and gr 1 to gr ½ t i d subsequently In cases of urgency digoxin should be given orally or intravenously The oral tablets contain *mg 0 25 The initial oral dose is mg 1 5 for a patient weighing* 10 stones or over and mg 1 to 1 25 for lighter patients Six hours later mg 0 25 is given and this is repeated every 6 hours until the ventricular rate falls to 70 or 80 Subsequently a maintenance dose of *mg 0 25* once or twice a day may be required In very urgent cases an intra venous injection of mg 1 of digoxin can be given 0 5 mg is put up in 1 ml of 80% alcohol This is diluted with nine times its volume of sterile saline and injected slowly Two hours later mg 0 25 of digoxin can be given by mouth Symptoms of overdosage of digitalis are undue slowing of the apex rate (below 60) coupling of beats due to a regular sequence of premature systoles the onset of pulsus alternans *diminution in the output of urine and vomiting* Vomiting at the onset of the treatment is usually due to congestion of the gastric mucosa, and not to the digitalis The urinary output should always be measured while the patient is taking *large doses of digitalis* In desperate cases strophanthin K gr 1/200 or ouabaine (a preparation of G strophanthin) gr 1/240 should be injected intravenously

*The Use of Quinidine* This may be given in selected cases with the object of restoring normal rhythm The main contra indications to the use of quinidine are myocardial degeneration a history of recent embolus heart failure or heart block The most suitable cases are those which are inflammatory in type and recent in origin or those occurring in association with hyperthyroidism It is usually considered inadvisable to administer digitalis and quinidine simultaneously The patient must be kept in bed A test dose of gr 3 (0 2 G ) of tab

quinidine sulph is given If no toxæmic symptoms appear such as sweating nausea vomiting diarrhœa abdominal pain a scarlatiniform rash dimness of vision or severe headache the administration can be proceeded with *On the next day gr 6 (0 4 G ) quinidine are given* every 3 hours for 4 doses and the day after gr 6 quinidine are given three hourly for 5 doses This may be continued for another 4 days The pulse and if feasible the apex should be examined before each dose is given and if found regular the quinidine should be discontinued The occurrence of frequent premature systoles is also an indication for omitting the drug If a regular rhythm is not restored at the end of a week on the above dosage the quinidine will probably have no effect, and should be stopped In cases in which the normal rhythm has been restored it may be necessary to give a daily dose of gr 5 for long periods When arterial embolism occurs the artery should be immediately incised and the clot removed

## Auricular Flutter

**Definition** A condition characterised by rapid and regular auricular contractions of a peculiar nature and almost invariably accompanied by heart block

**Etiology** Auricular flutter is usually associated with myocardial degeneration and arteriosclerosis much less frequently with infective diseases or with thyrotoxicosis

**Pathology** Prinzmetal has shown that flutter depends upon an irritable auricular focus The auricular rate is about 260 to 330 con tractions a minute and the ventricle usually contracts at half the auricular rate

**Clinical Findings** The patient is usually a male past middle age He gives a history of attacks of palpitations usually sudden in onset and in cessation but the last attack may have persisted

*On Examination* The heart is usually enlarged and the arteries are thickened A valvular lesion may or may not be present The pulse and apex rates are generally between 130 and 170 the rhythm is usually regular and the rate is unaffected by posture and by exercise Pressure on the carotid sinus will often slow the ventricular rate In the electrocardiogram regularly recurring dome shaped f waves are seen with ventricular complexes occurring at regular or irregular intervals but less frequently than the f waves owing to heart block

**Differential Diagnosis** Paroxysms of flutter can be differentiated from simple paroxysmal tachycardia by suitable tracings Auricular fibrillation may be simulated if in flutter the ventricular responses are irregular Slight exercise however in flutter usually renders the ventricular rhythm regular with a definite grade of heart block, such as 3 1 or 2 1 This does not occur in auricular fibrillation

**Course and Complications** Paroxysms of flutter may occur or a continuous stage may persist for as long as 10 years Paroxysms may ensue in which the ventricle assumes the auricular rate These are very dangerous the patient rapidly losing consciousness and dying Congestive heart failure may occur as a complication of flutter

**Prognosis** This is always serious, but not usually immediately grave The condition of the myocardium is a factor of great importance although hard to estimate

**Treatment** The patient should be put to bed and digitalised as for fibrillation This usually converts the flutter rhythm to that of fibrillation On stopping the digitalis a normal rhythm may ensue Quinidine alone should not be given until the flutter has been converted to fibrillation If given while flutter persists it should be combined with digitalis The normal rhythm may then be restored direct without an intermediate stage of fibrillation

### Ventricular Fibrillation

This is probably the cause of sudden death in coronary obstruction, *complete heart block diphtheritic myocarditis and following the* intravenous injection of mercurial diuretics It is compatible with life only if the fibrillation is of very short duration the clinical picture then *being that of Adams Stokes attacks An intracardiac injection of* Pronestyl 1 G should be given

### Heart block

**Definition** A condition characterised by delay in the conduction of impulses along some portion of the junctional tissue of the heart The following varieties are described 1 Sino auricular block 2 Auriculo ventricular block 3 Bundle-branch block 4 Arborisation block

**Etiology** Congenital heart block is due to a defect, such as a patent interventricular septum interrupting the bundle of His, or rarely to aorto pulmonary patency Acquired heart block may be due to inflammatory lesions caused by rheumatism diphtheria influenza pneumonia and enteric or scarlet fever or to degenerations, especially that caused by syphilis It may also result from overdosage with digitalis, strophanthin squills or quinidine A gumma tumour cyst or an area of fibrosis or atrophy may be the causative factor in some cases

#### *Sino auricular Block*

*The impulses arising in the S–A node at times fail to provoke an* auricular contraction The whole heart then misses a beat but the *succeeding contraction occurs at approximately the normal interval* The ventricle may occasionally interpolate a beat on its own (' ventricular escape ) when the heart is beating at the slow rate *It is a cause of dropped beats, and can only be distinguished from* auriculo ventricular block by a tracing the complete absence of the **P R T** deflections in the electrocardiogram corresponding with the pauses in the heart beats It is probably of no clinical importance but if occurring regularly every other beat the pulse is slow about 30 Exercise usually causes the rate to double and the restoration of normal rhythm can also be effected except in cases of long standing by the administration of atropin sulph gr 1/200 in 1 fl oz of water t i d

### Auriculo ventricular Block

Delay occurs in the passage of the impulse from the auricle to the ventricle Four grades may be recognised —

*Grade I* This is the earliest stage and can only be detected by a tracing The electrocardiogram shows a prolongation of the P–R interval to more than the normal of ⅕ second *Grade II* The ventricle occasionally fails to respond to the auricular impulse *Grade III* The ventricle fails in a regular manner to respond to the auricular stimuli Thus every fourth ventricular beat may be missing (4 3 block) or other sequences such as 3 2 or 2 1 block may be present *Grade IV* There is complete dissociation between auricle and ventricle the ventricle contracting regularly at its own independent rate of about 30 to 40 a minute The electrocardiogram allows all these grades of block to be diagnosed with certainty

**Clinical Findings** The patient does not usually notice anything abnormal except in the severe degrees of heart block Attacks of unconsciousness (Adams Stokes syndrome) are liable to occur if the A–V bundle fails to conduct the impulse and the ventricle does not take up its independent rhythm The attacks come on suddenly with convulsive movements of the face and arms usually the tongue is not bitten and there is no involuntary micturition The patient is pale and falls down he then becomes cyanosed and the breathing is stertorous Unless the ventricle begins to beat again he will die

*On Examination* Auscultation at the apex of the heart will show that the heart misses a beat when a beat is dropped at the radial pulse A ventricular rate below 40 a minute is almost always due to complete heart block It is often possible to see pulsation in the jugular veins corresponding in rate with the auricular contractions and so faster than the apex beat or radial pulse

**Differential Diagnosis** Clinically the missed beats which occur at the wrist in heart block must be distinguished from those resulting from feeble premature systoles In the former as described above there is silence on listening over the heart when the beat is absent at the wrist Inhalation of amyl nitrite in 2 1 heart block will often suddenly double the rate of the pulse the apex subsequently abruptly reverting to its slow rate This change does not occur in simple bradycardia The ventricular rate in complete heart block is not affected by exercise or by the inhalation of amyl nitrite or by the injection of atropine sulphate.

**Course and Complications** Heart block especially in the young when arising during the course of or in convalescence from an acute illness is usually a temporary derangement If due to a degenerative lesion it is likely to be permanent With the stage of onset of complete block the Adams Stokes syndrome is to be feared

**Prognosis** Heart block always indicates some degree of myocardial abnormality due either to an inflammatory lesion which may be temporary or to a permanent degenerative one The outlook is more serious when complicated by Adams Stokes attacks

**Treatment** When heart block develops during the course of an infective illness the patient should be kept strictly at rest in bed until

the normal rhythm is restored, or until it is considered that the heart block is permanent In other cases the Wassermann reaction should be determined and if positive a course of anti syphilitic treatment given (see p 596) For Adams Stokes attacks subcutaneous injection of m 8 of liq adrenal hydrochlor should be given immediately, followed by ephedrine sulphate gr ¼ t d s by mouth or isoprenaline sulphate (Neo Epinine) mg 10 tab sublingually Good results have been recorded in ventricular asystole by the intravenous injection of a molar solution of sodium lactate The solution is injected from a syringe taking ½ to 2 minutes The dose is 40 to 80 ml repeated if fresh episodes of asystole occur This may be a life saving procedure If the heart has stopped beating an intracardiac injection of m 8 of liq adrenal hydrochlor should be given

## Bundle branch Block

There is delay in conduction in one or other of the branches of the bundle of His, leading to the left or right ventricle This condition can only be diagnosed by means of an electrocardiogram In left bundle branch block the first heart sound may be split and in right bundle branch block the second heart sound may be split It is of grave significance death often occurring within three years of its detection but the prognosis depends upon the associated heart disease Right bundle branch block in an otherwise healthy individual is not of serious significance Adams Stokes attacks have also been described in bundle branch block

## The Wolff Parkinson-White Syndrome

The condition was originally described as bundle branch block but is now thought to be due to pre excitation or to accelerated conduction This means that the impulse instead of being delayed in its passage from auricle to ventricle is accelerated The impulse may pass along an abnormal pathway from auricle to ventricle which was described by Kent as the right lateral bundle thus reaching the right ventricle before arriving at the left ventricle Alternatively the impulse may travel along the normal path from auricle to ventricle but its passage through a portion of the A V node may be accelerated The Wolff Parkinson White syndrome is characterised electrocardiographic ally by (*a*) Shortening of the P R interval usually to 0 12 second or less (*b*) Widening of the QRS complex with slurring at its onset The P S interval is normal (*c*) Changes in the direction of the QRS complex with right or left axis deviation (*d*) Changes in the S T segment and T waves (*e*) Paroxysmal supraventricular tachycardia auricular extra systoles auricular flutter or fibrillation

**Clinical Findings** Paroxysmal tachycardia occurs in about 50% of the patients especially after exercise It generally shows itself before the age of 30 and males are more often affected than females It is usually benign but sudden death may occur

**Treatment** Quinidine in a dose of gr 5 by mouth may produce a

sudden and dramatic improvement Subsequently a small maintenance dose of quinidine such as gr 3 daily may be required

## Arborisation Block

The terminal subendothelial divisions of Purkinje s fibres have impaired conductivity This lesion can be detected by an electro cardiogram The prognosis is usually bad

## Pulsus Alternans

**Definition** Alternate strong and weak contractions of the ventricles

**Etiology** Pulsus alternans is probably a manifestation of myocardial degeneration the heart labouring against an excessive burden it may also result from an overdose of digitalis

**Clinical Findings** Pulsus alternans is difficult to detect by the finger A radial tracing will however show alternate large and small beats at very nearly regular intervals If the blood pressure is taken by the auscultatory method only alternate beats will be heard at higher pressures whereas on lowering the pressure in the armlet each beat will be audible An electrocardiogram may show R waves of equal intensity although the radial tracing shows definite alternation Pulsus alternans can be differentiated from premature systoles regularly recurring with every other beat (pulsus bigeminus) by the short interval between the normal and successive premature beat and the longer interval between the premature beat and the next normal one The condition is of grave import when occurring in a pulse of normal rate but if associated with paroxysmal tachycardia or with digitalis medica tion the prognosis is not so serious

## Pulsus Paradoxus

**Definition** A condition in which the pulse weakens in intensity with inspiration and becomes stronger with expiration

**Etiology** Pulsus paradoxus is often associated with mediastino pericarditis and with pericardial effusion and may be explained by the fact that normally during inspiration the roots of the lungs descend and carry the heart with them If however the heart is tethered by adhesions or pressed on by a pericardial effusion during inspiration it is pulled on from above and below The orifices of the venæ cavæ will then be partially obstructed with diminished cardiac inflow and output

# THE MYOCARDIUM

The pathological affections of the myocardium may be grouped as follows Atrophy Cloudy swelling Fatty degeneration and infiltra tion Fibrosis Amyloid hyaline and calcareous degenerations Granulomata Tumours simple and malignant Cysts Hypertrophy Dilatation Inflammation Vascular degeneration and infarct Rupture

*Atrophy* This is usually a brown atrophy It is common in old age and in wasting diseases such as tuberculosis and cancer *Cloudy swelling* This is met with in acute fevers and septicæmia *Fatty degeneration* This occurs in acute fevers diphtheria alcohol

chloroform or phosphorus poisoning and in blood diseases, suc anæmia and leukæmia It causes the change known as "tabby striation or "thrush s breast" appearance, seen under the endocard usually in the left ventricle *Fatty infiltration* This is sometimes not invariably, associated with obesity *Fibrosis* This is associ with arteriosclerosis and narrowing of the coronary arteries areas of fibrosis may be localised or diffuse and are comm found in the left ventricle It may occur diffusely as a sequel of r matic carditis *Amyloid hyaline and calcareous degenerations* T are less common *Granulomata* These include syphilis and tu culosis of the heart *Tumours* A lipoma or fibroma or a peduncul auricular myxoma may develop, or secondary melanotic sarc or rarely secondary carcinoma *Cysts* A hydatid may form in heart *Hypertrophy* This is associated with valvular disease, suc aortic or mitral regurgitation, with adherent pericardium increa blood pressure (hypertensive heart disease), thyrotoxicosis chr nephritis and arteriosclerosis The normal weight of the heart is 0 4% the body weight Congenital idiopathic hypertrophy is a rare condit which usually results in death in less than a year *Dilatation* This n be due to fatty degeneration to acute inflammatory changes or it n be associated with hypertrophy *Inflammation* Acute simple m carditis This occurs in infections especially rheumatic fever diphtheria and less often with enteric or scarlet fever septicæn Graves disease and syphilis Acute suppurative myocarditis Sn embolic abscesses may form in the myocardium in pyæmia as puerperal fever, osteomyelitis or malignant endocarditis *Vasc degeneration and infarct* These are especially associated with coron obstruction *Rupture* This may follow an infarct or result fr penetrating injuries, or occasionally from non penetrating injuries the chest wall

The following clinical varieties of myocardial affections will described The hypertensive heart the cor pulmonale the failing hea coronary obstruction and non penetrating injuries of the heart

The fatty heart and the fibroid heart do not justify separate clini descriptions Sudden death from fatty heart is due probably ventricular fibrillation, and not to the fat around the heart Fat degeneration of the heart however such as occurs in diphtheria leads cardiac dilatation tic tac rhythm and irregularities such as hea block or gallop rhythm It is a very serious condition Fibroid hea is often described as a condition of hypertrophy of the left ventric such as is found in hypertensive heart disease

### Hypertensive Heart Disease

**Definition** Hypertrophy of the left ventricle due to hyperpiesia

**Etiology** The cardiac enlargement is secondary to the high bloo pressure The cause of the high blood pressure (essential hypertensio is unknown but the subject is discussed on p. 281

**Pathology** There is hypertrophy of the heart The left ventric is chiefly affected The muscle fibres are increased in size

**Clinical Findings** The patient is usually an adult over the age of 40 He complains of symptoms due to high blood pressure (see p 281) No cardiac symptoms are noticed until the reserve of cardiac power is overtaxed or some complication such as disturbance of coronary circulation occurs Symptoms include shortness of breath on exertion attacks of cardiac asthma palpitations or præcordial pain

*On Examination* The heart The apex beat is usually displaced downwards and outwards A systolic murmur may be heard at the apex due to mitral regurgitation and occasionally there is an aortic systolic or diastolic murmur With failure of the heart pulsus alternans or presystolic gallop rhythm (see p 238) may be present The blood pressure which is raised in the early stages of the disease often falls with the onset of failure of compensation although the diastolic figure generally remains over 100 mm Hg Ophthalmoscopic examination may show retinal arteriosclerosis

**Differential Diagnosis** Hypertensive heart disease must be differentiated from cardiac enlargement associated with chronic nephritis or due to aortic disease or thyrotoxicosis The diagnosis is difficult in the later stages especially if the blood pressure has fallen

**Course and Complications** The course is progressive Complications include auricular fibrillation congestive heart failure cerebral hæmorrhage angina pectoris coronary thrombosis and rarely uræmia Intercurrent infections are not uncommon

**Prognosis** This is always unfavourable especially when there is marked cardiac enlargement retinal arteriosclerosis or myocardial failure

**Treatment** This is as described for hyperpiesia and for congestive failure (see pp 282 240)

## Endomyocardial Fibrosis

### (*Endocardial Fibro elastosis*)

Fibrotic thickening of the ventricular endothelium may occur as a congenital lesion or be associated with cardiac infarction or rheumatic carditis

In Africa where it is responsible for about a third of all cases of heart failure it is met with apart from these conditions The cause is not known it does not appear to be associated with malnutrition but it may result from sensitisation to streptococcal products There is thickening and fibrosis of the endocardium near the apex of the left ventricle and the mitral valve may also be thickened The right ventricle and tricuspid valve may also be affected

## Pulmonary Heart Disease

### (*Cor Pulmonale*)

**Definition** Enlargement of the right ventricle due to rise of pulmonary arterial tension

**Etiology** The normal pulmonary arterial pressure is between 18

and 30 mm Hg systolic and 7 to 10 mm Hg diastolic Rise of pressure in the pulmonary artery may be due to (1) Left ventricular failure and mitral disease (2) A diminution in the vascular channels in the pulmonary bed owing to such conditions as emphysema or fibrosis of the lungs (3) Loss of areas of lung tissue as occurs in tuberculosis, after resection of the lung etc (4) Obstruction of pulmonary arterioles by multiple emboli (5) Diffuse changes in the pulmonary arteries or arterioles resulting in pulmonary arteriosclerosis (6) Some varieties of congenital heart disease such as patent ductus arteriosus, and atrial and ventricular septal defects

When the pulmonary arterial pressure rises to about 60/30 mm Hg early signs of cor pulmonale appear

*Acute cor pulmonale* This may be due either to massive pulmonary embolism or to an attack of pneumonia occurring in a patient suffering from severe emphysema

*Chronic cor pulmonale* The more common causes are emphysema and bronchitis with spasm Other lung causes include pulmonary fibrosis pneumokoniosis, carcinomatosis schistosomiasis tuberculosis bronchiectasis and cysts of the lungs Among arterial causes are pressure by an aneurysm on the main pulmonary arteries pulmonary arteriosclerosis and cicatricial pulmonary arteritis Kyphoscoliosis is also a cause in some cases

Ayerza s disease is a term which some authorities consider should be abandoned The cyanosis (*cardiacos negros*) was due in the original case to long standing diseases of the lungs and was an example of chronic cor pulmonale

**Clinical Findings** In *acute cor pulmonale* the symptoms are those of massive pulmonary embolus dyspnœa distress and collapse being very marked

In *chronic cor pulmonale* the patient complains of progressively increasing dyspnœa followed by cough expectoration and sometimes hæmoptysis Lack of concentration giddiness palpitations and somnolence may also be noticed

*On Examination* In *acute cor pulmonale* there is cyanosis with venous engorgement in the neck The pulse is very feeble and rapid and the dyspnœa intense The pulmonary second sound is accentuated and protodiastolic gallop rhythm may be present The patient usually rapidly loses consciousness and dies In *chronic cor pulmonale* signs of right sided cardiac failure are present cyanosis dyspnœa tachycardia and low blood pressure The heart dulness is enlarged to the right and left if it is not obscured by emphysema The increase in dulness is due to the right auricular dulness extending to the right, and the right ventricular dulness to the left A systolic murmur is sometimes heard over the tricuspid area An electrocardiogram will show right axis deviation with tall P waves and inverted T waves in leads II and III and an X ray examination will reveal the enlargement of the right auricle and right ventricle In more advanced cases the veins of the neck are engorged the liver enlarged tender and pulsating and there is œdema of the legs A transudate may form in the pleural sacs usually more

on the right side and there may be hydropericardium Recurrent attacks of papilloedema may be noted varying with the degree of heart failure

**Treatment** *Acute cor pulmonale* The patient should lie propped up and oxygen should be administered through a B L B mask or polymask Removal of a pint of blood from a vein in the arm may afford considerable relief Ten ml of Coramine or mg 1 of strophanthin K may be injected intravenously or an intravenous injection given of 0 24 G Cardophylin in 10 ml sterile water

*Chronic cor pulmonale* Oxygen should be administered cautiously as described under emphysema on p 172 The heart failure should be treated with digitalis mercurial diuretics and a low salt diet Pulmonary infection and bronchospasm should be treated as described in the section dealing with respiratory disorders Morphine should not be administered but rest and sleep may be secured by the administration of paraldehyde m 60 to 120 by mouth in m 30 capsules or by an intramuscular injection of paraldehyde 5 to 10 ml (10 ml ampoules)

## Primary Pulmonary Hypertension

This is a rare disease in which there is obstruction of the smaller pulmonary arteries by endarteritis fibrosa The cause is unknown Adults and children may be affected The patient complains of lassitude dizziness fainting attacks pains in the chest and shortness of breath Cyanosis is often marked with polycythæmia The extremities are pale There is enlargement of the right side of the heart and the electrocardiogram shows right ventricular hypertrophy Cardiac catheterisation reveals a rise of pressure in the right ventricle to perhaps 50 mm Hg (normal about 25 mm Hg )

## The Failing Heart

### (*Congestive Heart Failure*)

**Pathogenesis** The exact nature of heart failure is still uncertain The back pressure theory was generally accepted in the middle of the last century According to this left ventricular failure causes dilatation and incompetence of the mitral valve with resultant congestion of the lungs and subsequently, secondary failure of the right ventricle and dilatation of the tricuspid valve lead to systemic congestion. Later, the forward theory was introduced heart failure being considered as due essentially to inefficiency of the driving force of the heart muscle with in consequence deficient blood supply to the tissues This theory was largely based on observations made on heart failure in auricular fibrillation which often affects both sides of the heart simultaneously A return was then made to the older view that the symptoms of heart failure are mainly due to congestion occurring in the circulatory territory immediately behind the side of the heart which first fails Observations made by cardiac catheterisation indicate that in congestive heart failure there is a rise in venous pressure which results in over

loading of the heart Further rise in the venous pressure resul diminished cardiac output Heart failure may thus affect either of the heart independently, or both sides may be involved simul ously Further, such failure may be acute or insidious in onset what has been said above, it will be clear that we should expect to systemic congestion with right sided failure and pulmonary conge with left sided failure

**Etiology** 1 *Left heart failure* This is approximately three t as common as right heart failure The important causes are hy tensive heart disease, aortic stenosis aortic regurgitation chi nephritis and coronary occlusion 2 *Right heart failure* This is frequently secondary to left heart failure Other important causes been mentioned in the article on Pulmonary Heart Disease Bernheim s syndrome or isolated right heart failure there is syst venous engorgement without pulmonary congestion It is associ with hypertrophy and dilatation of the left ventricle due to hy tension, kidney disease or combined aortic and mitral valvular lesi The patient dies with the classical features of right sided failure an autopsy the right ventricle is found to be stenosed as the resul encroachment in the right ventricular space of the hypertropl interventricular septum The arm to tongue circulation time generally normal and despite the raised venous pressure and œde the patient can often lie flat in bed without distress 3 *Left and r heart failure* This may be associated with rheumatic carditis diphthe severe anæmia generalised coronary arteriosclerosis auricular fibri tion auricular flutter, paroxysmal tachycardia hyperthyroidism constrictive pericarditis

Heart failure may be acute or insidious for example, acute heart failure may follow hypertension aortic disease or coron occlusion, and acute right failure may be associated with pulmon embolus thrombosis or lobar pneumonia

**Clinical Findings** *Left heart failure* Three stages are describ (*a*) Paroxysmal pulmonary congestion Attacks of acute œdema the lungs or of nocturnal dyspnœa occur, with cough and lesser degr *of pulmonary œdema* (*b*) *Pulmonary congestion of effort* H shortness of breath cough and often blood stained sputum are provok by exercise excitement or a cold atmosphere (*c*) Chronic pulmona congestion This is characterised by shortness of breath and at tim hæmoptysis Such cases are liable to be mistaken for chronic bronchit pulmonary tuberculosis or a new growth

The chief clinical features of left heart failure are pallor with so cyanosis and dyspnœa especially on effort and at night The heart usually enlarged to the left and gallop rhythm or pulsus alterna may be present (see p 233) With gallop rhythm a third heart sou is heard during mid or late diastole the heart sounds are then liken to the sound produced by saying lub lub dupp Graphic recor indicate that the first part of the first sound occurs in presystole and presumably auricular in origin It is also known as presystolic gall rhythm the extra sound perhaps being due to vibrations of a rapid

filling ventricle which lacks tone It does not occur in auricular fibrillation This third sound can also be felt as a diastolic impulse The three heart sounds are evenly spaced and the third sound is only heard when the rate is rapid It is of very grave significance Presystolic gallop must be distinguished from protodiastolic gallop rhythm In the latter a third sound closely follows the second sound lub dupp dupp ' This is of no significance being an accentuation of the physiological third heart sound It is best heard near the apex of the heart whereas a split second sound is most noticeable near the base The blood pressure is usually high especially the diastolic figure and the rhythm is usually regular but premature systoles or paroxysmal auricular fibrillation may occur Rales are heard at the bases of the lungs and a hydrothorax may develop more frequently on the left side and if bilateral it is usually larger on the left side It is sometimes interlobar The electrocardiogram often shows left axis deviation with inverted $T_1$ or $T_1$ and $T_2$ waves or there may be bundle branch block X ray examination shows that the pulmonary arterial shadows are unduly dense and enlarged and a blurred zone around the pulmonary roots indicates the onset of pulmonary œdema If right sided failure now supervenes the symptoms of pulmonary congestion are to a certain extent relieved and paroxysms of dyspnœa usually cease The attacks of nocturnal dyspnœa are often known as *cardiac asthma* and they may be associated with bronchial spasm The patient wakes up suddenly feels suffocated sits up struggles for breath and finally he may fall back exhausted and sweating The attacks may be more severe the patient having acute œdema of the lungs Cheyne Stokes breathing is seen in other cases the breathing waxing and waning with intervals of apnœa lasting 30 or 40 seconds During the hyperpnœic period which results from stimulation of the respiratory centre $CO_2$ is washed out of the blood This produces the apnœic phase The patient may sleep in the apnœic phase and wake with each period of hypernœa

*Right heart failure* This has been described under Pulmonary Heart Disease

The determination of the arm to tongue circulation rate and of the venous systemic blood pressure affords further evidence of left or right ventricular failure With left ventricular failure the arm to tongue circulation time is increased but the systemic venous pressure is usually normal whereas with right ventricular failure both the circulation time and the venous pressure are increased.

*The circulation rate* The arm to-tongue circulation rate is used to determine the rate of circulation through the lungs Five ml of a 20% solution of Decholin (sodium dehydrocholate) are injected through a wide bore needle into an antecubital vein of the right arm the injection taking 2 to 3 seconds The patient sits propped up at an angle of 45 and a local anæsthetic is used to prevent tachycardia resulting from pain of the injection The patient is told to raise his left hand immediately he notices a bitter taste under the tongue The time is taken with a stop watch from the beginning of the injection of the Decholin to the signal that it has reached the tongue The normal time is between

10 and 17 seconds and, with both right and left heart failure, it m be increased up to 40 seconds or more

*The systemic venous blood pressure* This can be gauged clinica by observing the cervical veins with the patient recumbent and propp up Normally the venous pressure is zero at the point in the ve which is in the same horizontal plane as the lower end of the sternu With an increase of venous pressure the cervical veins on both si will be engorged at a point which is on a plane higher than that of t manubrium zero level An increased systemic venous pressure is almo certainly present if the cervical veins are dilated when the patient propped up at 45° The systemic venous blood pressure can be measur directly by inserting a short, bevelled 1 mm bore needle into an ant cubital vein of the right arm with the patient propped at an angle of 4! The needle is connected by a short piece of rubber tubing to the ho zontal arm of an L shaped graduated glass tube of 8 mm bore T whole apparatus is sterilised and a sterile 8 8% sodium citrate soluti run through it immediately before use The blood enters the glass tu and a reading is taken of the height of the column of blood above t level of the 4th intercostal space The normal reading is about 6 cı of blood Compression of the upper arm should cause the level to ris and on releasing the pressure it should fall to approximately its previo level indicating that there is no block in the apparatus

*Catheterisation of the Right Auricle* Catheterisation of the rigl auricle, by inserting the catheter into the basilic vein in the left ar and passing it into the right auricle has indicated that heart failu may occur either with a high or a low output The normal cardi output is 5 litres a minute In severe anæmia the cardiac output ma reach 12 to 14 litres a minute the venous pressure then rising A hig output also occurs in emphysema and in Paget's disease, owing to th highly vascular state of the bones in the latter condition Heart failui in valvular arteriosclerotic and hypertensive heart disease is associate with an output as low as 8 litres a minute Digitalis lowers the venou pressure and increases the cardiac output in low output failure In hig output failure digitalis by lowering the cardiac output does not appea likely to produce clinical improvement

Treatment *Acute Left Heart Failure* If there is œdema of th lungs an injection of morphin sulph gr 1/8 and atropin sulph gr 1/5 should be given This should be followed by venesection of 15 to 20 oz Digitalis in large doses is indicated in other cases or to produce a rapid effect an intravenous injection of strophanthin gr 1/200 or o ouabain mg 1 should be given

*Acute Right Heart Failure* This has been described on p 237

*Routine Treatment of Heart Failure* The patient should be in bed, on absolute rest propped up in the most comfortable position He may obtain relief by leaning forward on a well padded heart table, placed across the bed Diet The meals should be taken dry and not more than 80 oz of fluid drunk in the 24 hours The total calorie value should be low, 800 to 1,000 calories A specimen diet of 810 calories is as follows —7 a m Tea 1 cup with milk 1 oz and two level

teaspoonfuls of sugar Breakfast One egg (lightly boiled) Bread or toast 2 slices Butter ½ oz Jam or honey ½ oz 11 a m Water 8 oz Lunch Fish 2 oz Green vegetables 2 oz Bread 1 slice Butter ½ oz 4 p m Tea 1 cup with milk 1 oz and two level teaspoonfuls of sugar 6 p m Water 8 oz Dinner Toast 1 slice Jam or honey ½ oz Junket 6 oz No salt should be taken The amount of urine passed every 24 hours should be measured and charted Digitalis should be given as tab digital præp gr 3 t d s on the first day gr 2 t d s on the second day and gr 1 t d s or less often subsequently keeping watch for the onset of toxic symptoms such as nausea vomiting oliguria bradycardia or coupled beats Digitalis may be given if the patient is suffering from heart block Mersalyl should also be given to increase diuresis It is administered as follows A test dose of 0 5 ml is injected intramuscularly. If there is no intolerance the signs of which are hæma turia diarrhœa or cutaneous irritation 2 ml of mersalyl are injected intramuscularly twice a week and ammon chlorid 2 G is given t i d the day before and the day of the injection The ammon chlorid is put up in 0 5 G (gr 7½) enteric coated tablets or as Emplets If diuresis is not provoked after the second injection it is not usually likely that the treatment will succeed Cation exchange resins combined with a low salt diet may also be used for the treatment of cardiac œdema (see p 478) Cardophylin can also be used as a diuretic and to dilate the coronary vessels It can be given by mouth as a tablet (0 1 G ) two to four daily or as an intravenous injection of 0 24 G in 10 ml daily, or as an intramuscular injection of 0 48 G in 2 ml daily A hydrothorax should be aspirated as it severely impedes the heart s action similarly ascitic fluid should be removed by drainage if it does not show signs of absorption If the œdema persists in the legs it is advisable to sit the patient up in a heart chair for 12 hours and then drain the legs by making multiple small incisions ¼ inch long through the skin which has been sterilised with ether on the dorsum of the feet and lower part of the front of the legs The legs are then covered with sterile gauze which is changed as it becomes soaked with the fluid the legs being further protected by a cradle covered with a blanket The gauze usually requires changing every 3 or 4 hours Venesection should be performed for right sided engorgement and oxygen administered Sleep should be secured either by the use of chloralamide in a mixture such as Chloralamid gr 30 pot brom gr 15 sp chlorof m 20 aquam ad fl oz 1½ fl oz 1½ nocte by paraldehyde m 120 in gin fl oz 1 by mouth by the rectal injection of paraldehyde fl oz ¾ to 1 in fl oz 4 of warm saline by Nepenthe m 15 and aspirin gr 10 or by a sub cutaneous injection of morphin sulph gr 1/6 to 1/4 with atropin sulph gr 1/120 nocte For cardiac asthma an injection of morphine should also be given Whisky fl oz ½ to 1 will often relieve restlessness The bowels should be kept open freely with the help of salines such as mag sulph gr 60 to 120 mane A total thyroidectomy has produced good results in some cases of congestive failure not associated with hyperthyroidism which have failed to respond to other measures Alternatively thiouracil may be given in doses of mg 200 3 or 4 times

a day for 3 or 4 weeks Mersalyl and ammonium chloride are of great value in cases of pulmonary congestion of effort and in chronic pulmonary congestion, where the pulmonary congestion may be latent

*The After Treatment* The patient must be kept in bed until the pulse rate remains steady at about 80 He may then be allowed to sit out of bed for half to one hour daily, and subsequently gradually walk about on the level The fluid intake should be restricted to 30 to 40 oz in the 24 hours and a weekly injection of mersalyl with the administration of ammon chlorid 2 G t i d for three days a week may be of great benefit During this period of convalescence massage to the extremities and the body is of great value The walking exercise should be carefully regulated and the patient should sit down immediately he experiences any cardiac distress

## Peripheral Circulatory Failure

The circulation may fail primarily in the periphery rather than in the heart This is due to slowing of the circulation caused by dilatation of the arterioles so that there is failure of the venous return to the heart and diminished cardiac output It is met with typically in surgical shock, but may occur in acute infections such as diphtheria or pneumonia The blood pressure is very low The pulse is regular but very feeble the skin is pale and cold but the heart beat is forcible In pneumonia the blood may be pooled in the skin giving rise to cyanosis Treatment of peripheral failure is often unsatisfactory Ephedrine hydrochlor gr ½ should be given by mouth and Pitressin 1 ml (20 units) injected intramuscularly every 6 hours, and an intramuscular injection of Coramine 1 5 ml given t i d The foot of the bed should be raised and hot bottles applied to the patient s feet

## Syncopal Attacks

**Definition** Unconsciousness due to deficient cerebral circulation

**Etiology** The circulatory failure may be primarily vascular due to deficient supply of blood to the heart, or primarily cardiac due to deficient output from the heart

The chief causes are thus anæmia, low blood pressure change of posture, vaso vagal attacks an over active carotid sinus reflex heart block and auricular flutter

**Clinical Findings** The clinical findings are described under the respective headings of the causative conditions Postural syncope does not occur when the patient is lying In all syncopal attacks, as opposed to fainting attacks of cerebral origin and not due to disordered cerebral circulation the pulse is weak An unduly sensitive carotid sinus reflex results in reflex vagal stimulation and slowing of the heart. It occurs especially with a raised blood pressure and arteriosclerosis Pressure over one or other carotid sinus may slow the heart and produce an attack of syncope in those predisposed

**Treatment** This is described under the respective headings A hypodermic injection of atropin sulph gr 1/100 or of m 5 of liq adrenal hydrochlor will usually relieve vaso vagal attacks For carotid sinus syncope ephedrine sulph gr ¾ t d s by mouth may

afford relief and in severe cases denervation of the sinus has been performed

### Ischæmic Heart Disease

This is the name sometimes given to the condition associated with deficient circulation through the coronary arteries It may result from various causes such as aortic stenosis atheroma of the coronary arteries thrombosis of the smaller branches of the coronary arteries without infarct formation anæmia and thyrotoxicosis In old age areas of fibrosis may develop in the myocardium

## Angina Pectoris

**Definition.** A condition characterised by paroxysmal attacks of substernal pain of grave prognosis and often associated with changes in the aorta heart or coronary arteries

**Etiology** It is now generally believed that in angina the pain impulses arise in the heart muscle as the result of interference with its blood supply Myocardial ischæmia produces pain possibly owing to anoxia, possibly as the result of retention of a pain producing factor liberated by muscular contraction and retained locally owing to deficient circulation The deficient blood supply may be due to disease obstruction or spasm of the coronary arteries Further in some cases anginal symptoms are associated with severe anæmia diabetes mellitus spontaneous hypoglycæmia hyperthyroidism and hypothyroidism Angina is not however a regular accompaniment of these diseases, there must therefore be some other causative factor than diminished oxygen or sugar supply to the heart muscle impaired coronary filling or low muscle metabolism The additional factor in those cases in which angina is present may be coronary narrowing due to lesser degrees of disease or spasm *Exciting causes* include muscular exercise which is the characteristic instigator of *angina of effort* and exposure to cold mental exertion and excitement a heavy meal and possibly over indulgence in tobacco which lead to coronary spasm and *spasmodic angina* Angina is often associated with syphilis but it may follow an acute illness such as influenza malaria or rheumatic fever It tends to run in families and is more common in men especially after the age of 50 Brain workers are liable to angina

**Pathology** The coronary arteries are usually diseased showing either calcification endarteritis or occlusion of their orifice by aortitis or atheroma of the aorta The aorta is often affected syphilitic aortitis atheroma or aneurysm may be present The myocardium is frequently diseased If the patient dies in an attack the heart is usually relaxed and full of blood Ischæmic fibrosis is often found resulting from obliterative arteritis of the coronary vessels

**Clinical Findings** The patient is usually a man over the age of 50 Angina does rarely affect women and young people may suffer from it as the result of syphilis or an acute illness such as influenza In *angina of effort* the patient complains of attacks of pain which have usually a sudden onset and cessation and are provoked by exertion

which may, however, only be of a very slight degree such as walking a *few yards, or in more severe cases,* turning over in bed or talking The characteristic feature is that in each case there is a direct and often a quantitative relationship between exercise and the onset and the severity of cardiac pain Further, with rest the pain is rapidly relieved Attacks of *spasmodic angina* occur apart from exercise Further angina of effort and spasmodic angina may occur simultaneously or consecutively The attack of spasmodic angina does not cease with *rest and tends to run its course unless* relieved by drugs An attack of spasmodic angina may be induced by mental excitement such as that provoked by hearing an air raid siren by exposure to cold, or by going into a cold room or by the cold sheets in a bed, a heavy meal is at times provocative The pain usually begins in the mid line behind the sternum and may radiate thence to the left shoulder down the ulnar side of the left arm, to the left side of the neck and jaw, or to the scalp I have *seen a case in which it was only felt in the jaw, in others it* remains localised to the back or wrist Often the pain radiates to the right side also In some instances the pain is first felt in the region of the xiphisternum or epigastrium (abdominal angina), or in the arteries of the arm The pain is very severe continuous while it lasts and non throbbing A sense of constriction, as if the chest were held in a vice, is also felt simultaneously with or shortly after the pain This is probably due to contraction of the intercostal muscles The attack is sometimes accompanied by severe mental anguish and by a sensation of impending death This is more likely to occur with spasmodic angina The attack may last for only a second or for several minutes in some instances a 'status anginosus ensues in which a series of attacks rapidly follow one another Angina sine dolore is also described, in which the patient is suddenly seized by a sensation of imminent death becomes pale and motionless and yet experiences no pain During an attack salivation or vomiting may occur the attack ceasing with eructation of wind or a copious flow of urine Minor attacks vary in severity from slight substernal distress on exertion to definite pain

*Examination of the Patient During the Attack* He is usually pale motionless and silent he may however be flushed or groan Frequently the pulse is unaffected but at times it is feeble The blood pressure usually rises and may reach a figure of 340 mm Hg syst in some instances it is not affected Sudden death may occur during the attack perhaps from vagal inhibition or from ventricular fibrillation The heart may show no abnormality clinically or there may be enlargement When the examination is made between the attacks, cardio vascular degeneration is usually found with arteriosclerosis and increased blood pressure, and shortly after an attack tender spots are often *present over the præcordium or along the arm* The electro cardiogram between the attacks is usually normal During or immediately after an attack or if the patient exercises sufficiently to produce pain the electrocardiogram shows in about 90% of cases, depression of the RS–T segment in the majority of leads

**Differential Diagnosis** Angina must be differentiated from 1 Left inframammary pain 2 Coronary thrombosis 3 Intercostal pain from other causes 4 Biliary and intestinal colic 5 The scalenus anticus syndrome 6 Diaphragmatic hernia 7 Oesophageal spasm

1 *Left inframammary pain* The pain is usually a dull ache but it may be very severe It is situated near the apex of the heart and may radiate to the left arm or shoulder It is present apart from exercise but is frequently accentuated by exercise The patient is often a woman suffering from a chronic anxiety state In addition to the pain she may complain of feeling tired of attacks of sighing respiration trembling and sweating No sign of disease of the heart or arteries can be detected

2 Coronary thrombosis The differential diagnosis is considered on p 248

3 Intercostal pain from other causes includes a consideration of cervical spondylosis dorsal spinal arthritis neuralgia, myalgia pleurisy, loculated spontaneous left sided pneumothorax and herpes zoster

4 Biliary and intestinal colic These conditions should present no *difficulties in differential diagnosis if full investigations are carried out*

5 The scalenus anticus or thoraco axillary inlet syndrome (see also p 448) This is due to pressure of a cervical rib of a rudimentary first rib or a normal first rib or of the scalenus anticus muscle on the brachial plexus The pain is neuralgic in character It may spread from the neck down the arm to the hand and is increased by rotation of the head to the affected side and by a downward pull on the shoulder Præcordial pain may also be present

6 Diaphragmatic hernia The diagnosis is established by a barium examination of the œsophagus and stomach in the erect and Trendelen berg positions

7 Œsophageal spasm The barium swallow will establish the diagnosis

***Course and Complications*** *In some cases the patient dies during* his first attack of angina in others the attacks recur with gradually increasing severity and progressive myocardial weakness Complica tions include acute œdema of the lungs cerebral hæmorrhage or throm bosis coronary thrombosis may follow previous attacks of angina

**Prognosis** This is always very uncertain but sudden death occurs in about 60% of cases and of the remainder a few recover whilst others have repeated attacks at intervals up to 20 years before dying either from angina from heart failure or intercurrent disease Unfavour able signs are attacks which are provoked by very slight exertion attacks which occur during the night and the presence of pulsus alternans indicating severe myocardial degeneration

**Treatment** *During the Attack* The immediate indication is to relieve pain A tablet of trinitrin gr 1/130 should be dissolved under the tongue or a capsule of amyl nitrite (3 to 5 minims) should be broken and inhaled through the nose This almost invariably gives relief if the blood pressure is raised One or two ounces of brandy or whisky frequently relieve the pain if amyl nitrite is not available If this fails a subcutaneous injection of morphin sulph gr ¼ should be given

or an inhalation of chloroform A subcutaneous injection of atropin sulph gr 1/100 should also be given with a view to overcoming vagal inhibition which may cause death If there is much flatulence the patient should be given m 60 of sal volatile (sp ammon aromat B P) with an equal quantity of water The tenderness over the sternum may be relieved by the application of hot flannels

*Between the Attacks* If the angina is a new symptom or if the attacks are becoming more frequent the patient should go to bed for a month and be given a course of anticoagulants (see p 186) In the majority of cases of angina it is not necessary for the patient to take to his bed The pain can often be warded off by a trinitrin tablet before the patient goes out in the morning or by taking a Cardophylin tablet gr 1½ t i d Peritrate has a more prolonged action than nitroglycerin It is given by mouth mg 10 tab 1 or 2 before meals and at bedtime Smoking should be given up if it is found that it is a causative factor Alcohol in moderation is not harmful Total thyroidectomy has now been generally abandoned Thiouracil has been used in angina 200 mg t d s with some measure of success the patient being free from attacks while under treatment In a series of cases so treated the basal metabolic rate varied from + 32 to − 9 at the beginning of treatment but in cases showing a favourable response the B M R fell during treatment If the result is good the thyroid activity can now be suppressed by radio iodine For the nervous excitability which is so frequently present ammon brom gr 10 or phenobarbitonum gr ½ may be given t i d Angina due to anæmia, diabetes mellitus hyperthyroidism and hypothyroidism requires treatment of the associated disease together with the administration of sedatives or vaso dilators p r n

Jonnesco's operation consisted in the resection of the whole of the cervical sympathetic and first thoracic ganglion In some cases stellate and upper dorsal ganglionectomy are now performed with relief of pain Promising results have been obtained by the operation of cardio omentopexy A portion of the omentum is brought through the left diaphragm and sutured to the edges of the divided pericardium and to the surface of the heart The blood supply of the ischæmic heart muscle is thus increased

### Cardiac Infarction

*(Coronary Thrombosis Coronary Occlusion)*

**Definition** Occlusion of a branch of a coronary artery with associated cardiac infarction and not infrequently a localised pericarditis ✓

**Etiology** Cardiac infarction is usually due to thrombosis in an atheromatous coronary artery rarely to syphilis causing a narrowing of the orifice of the coronary arteries or to an embolus in infective endocarditis

The view that coronary atherosclerosis is due to excessive fat in the diet causing an increase in the serum cholesterol has been emphasised and supported by observations on the dietary habits and incidence of coronary heart disease in Europeans Cape Colonials and Bantus in the Cape Peninsula On the other hand we see many patients with a high blood cholesterol level who do not suffer from coronary thrombosis and

during the war when the fat intake in this country was low the incidence of coronary occlusion did not appear to fall

**Pathology** The descending branch of the left coronary artery is most often affected an infarct forming in the left ventricle Softening fibrosis or calcification may develop in the infarct There is usually a localised fibrinous pericarditis over the infarct and aneurysm or rupture of the heart may ensue

**Clinical Findings** The patient is usually a male over the age of 50 *He may give a history of previous short attacks of anginal pain* In a typical case he is suddenly seized while at rest with severe pain in the middle or lower part of the sternum The pain may spread to one or both arms to the neck jaw or abdomen In addition there may be severe dyspnœa, nausea and vomiting The pain is persistent and lasts for several hours or for a day or so

*On Examination* The patient is usually restless pale and sweating and perhaps cyanosed The heart may show no abnormality at the onset but the sounds may be distant or an abnormal rhythm may be present The pulse This is often weak, and the rate 90 or 100 The blood pressure characteristically falls a systolic pressure of 100 mm Hg or even 80 being recorded In some cases there is evidence of venous engorgement with swelling of the jugular veins cyanosis and enlargement of the liver A few cases have been observed in which there was an absence of pain the patient being suddenly seized with severe dyspnœa in others the onset is syncopal with loss of consciousness The temperature rises shortly after the onset and may remain raised for 3 or 4 days this may be demonstrated in some cases by taking the rectal temperature which is above normal when the mouth temperature may show no rise A leucocytosis of about 20 000 per c mm is generally present with an increase in the sedimentation rate of the red cells A pericardial rub often develops in a day or so serving to confirm the diagnosis Anuria occurs when there is grave shock The diagnosis can usually be established by the electrocardiographic findings In a few cases no changes are present at the onset but typically soon after the onset there is a deviation of the R–T segment usually in opposite directions in leads I and III Lead IVR is of value in suspected cases of coronary occlusion In some cases it may show changes usually elevation or depression of the R–T segment with changes in the T wave when the three conventional leads are inconclusive

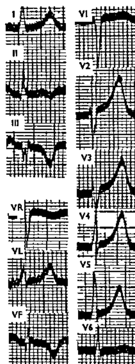


Fig 14 Electrocardiogram showing unipolar leads in posterior infarction VF shows elevation of RS–T segment and the sharply inverted T wave Lead III shows similar changes

Later, the T wave is inverted in leads I or III ($T_1$ type or $T_3$ type) and during convalescence the electrocardiogram is gradually restored to normal The unipolar leads enable a more exact localisation of myo cardial infarcts to be made and afford a more definite diagnosis in cases of suspected posterior infarcts (see Fig 14)

**Differential Diagnosis** Coronary thrombosis is differentiated from angina pectoris on the following grounds In coronary thrombosis the onset usually occurs when the patient is at rest He becomes restless the pain is of long duration and dyspnœa is often present, with sweating The pulse is feeble the temperature rises the blood pressure falls the cardiac rhythm may become abnormal and pericarditis ensue there is a leucocytosis, an increased sedimentation rate of the red cells the electrocardiogram is typical, and the pain is not relieved by nitrites In angina on the other hand, the onset is usually related to exercise the patient stands still the pain is of brief duration there is no dyspnœa, and usually little sweating, the pulse is not feeble the temperature does not rise the blood pressure rises, the cardiac rhythm is undisturbed there is no pericarditis or leucocytosis and the sedimentation rate is not increased The electrocardiographic changes are not so marked and relief is given by nitrites An acute abdominal condition may be simulated when the pain is referred to the abdomen and there is vomiting and collapse Other causes of severe dyspnœa require exclusion in the painless type of coronary thrombosis A dissecting aneurysm slowly rupturing into the pericardium may exactly simulate an attack of coronary thrombosis but no typical electrocardiographic findings are obtained

**Course and Complications** The patient may die immediately, or complications such as congestive heart failure pericarditis cerebral thrombosis or embolus venous thrombosis in the legs and pulmonary embolus may ensue Cardiac aneurysm and rupture of the heart are rare complications Repeated attacks are not infrequent

**Prognosis** About one third of the cases of coronary thrombosis are immediately fatal Another third die during the first six weeks of treatment in hospital The mortality rate of those treated on modern lines with anticoagulants has been reduced to about 15% A second *attack occurs in less than half the cases which survive the first attack* A few survive for periods up to twenty years but the average expecta tion of life is about eight years

**Treatment** Nitrites must not be given If the patient is collapsed with a low blood pressure he should lie flat if there are signs of cardiac failure he should be propped up An immediate subcutaneous injection of morphin sulph gr ¼ to ½ is required and this is repeated if necessary to relieve pain up to gr 1 in 4 hours Phenobarbitone is given in doses of gr 1 twice or three times daily If there is serious congestive heart failure digitalisation should be effected but it should be remembered that digitalis strophanthin and adrenaline may produce ventricular fibrillation The latter should never be given and the dosage of digitalis must be carefully watched Oxygen administered through a nasal catheter or B L B mask may help to relieve pain restlessness and

dyspnœa A hot water bottle should be placed near the feet For severe shock with a blood pressure of 50 mm Hg or less 1 noradrenaline tartrate (Levophed) should be given by intravenous drip The dose is 4 ml of 1/1 000 noradrenaline in a litre of 4 3% dextrose in 0 18% saline The amount of fluid injected must not overload the circulation and the blood pressure should be raised to 100 mm Hg It may therefore be necessary to increase the strength of the noradrenaline to 8 or even 64 ml per litre Frequent blood pressure readings should be taken Very little food should be given during the first few days of the illness and subsequently a diet of about 800 to 1 000 calories is advisable (see p 240) The fluid intake should be restricted to 30 to 40 oz No aperient should be administered for a week when an enema may be given if required The use of anticoagulants (see p 186) for 3 to 4 weeks has reduced the death rate and the occurrence of thrombo embolism by about 50% Anticoagulants should not be given if there is hepatic or renal disease severe anæmia pregnancy or in the presence of a peptic ulcer or of diverticulitis The patient should be kept in bed for at least a month from the onset of the illness

### Non penetrating Injuries of the Heart

The heart may be damaged by trauma to the chest wall without the ribs or sternum being fractured Lesions may occur in the pericardium myocardium or endocardium Fibrinous pericarditis pericardial effusions or adherent pericardium have been noted The heart may rupture or an area of scar tissue with subsequent aneurysm of the heart may develop Injury to the mitral valve has been followed by stenosis If the blow is severe immediate death may result from ventricular fibrillation In other cases there is collapse followed by præcordial pain tightness of the chest dyspnœa and palpitations The physical signs vary with the lesion produced Abnormalities are likely to be present in the electrocardiogram The T waves may be large or inverted and the R T interval elevated or depressed Typical coronary T waves may be seen with slurring of the QRS complexes The patient should be treated as if suffering from coronary occlusion

## THE ENDOCARDIUM

### Acute Bacterial Endocarditis

*(Malignant or Ulcerative Endocarditis)*

**Definition** An acute progressive bacterial inflammation of certain parts of the endocardium accompanied by embolic manifestations

**Etiology** There is usually an old valvular lesion A focus of infection is present in some part of the body from which organisms are carried to the heart The focus is often latent and may be in the intestines tonsils teeth or elsewhere Malignant endocarditis may also be associated with pneumonia osteomyelitis otitis media typhoid or scarlet fever and diphtheria More rarely a primary form occurs in which no extraneous septic focus can be discovered and in which there

is no evidence of a previous valvular lesion The organisms multiply in the heart The organisms are met with in the following order of frequency, the streptococcus hæmolyticus the staphylococcus aureus, the bact coli, the pneumococcus the gonococcus the meningococcus Rarely the infecting organisms are the Hæmophilus influenzæ the streptococcus viridans the staphylococcus albus etc This disease has become comparatively rare since the use of antibiotics

**Pathology** The valves and the endocardium lining the cardiac chambers are chiefly affected Vegetations form on the surfaces of the valves which are in apposition, such as the auricular aspect of the mitral valve segments and the ventricular surface of the aortic valve cusps The left side of the heart is usually affected but in cases of congenital heart disease vegetations form on the right side They constitute excrescences from the valve or spread along the posterior wall of the left auricle the wall or septum of the left ventricle (mural endocarditis) or along the aorta Deep seated erosions often occur and in this way a valve cusp, the interventricular septum or the heart wall may be perforated or the chordæ tendineæ ruptured Septic infarcts are liable to form in the spleen kidneys brain, intestines retinæ, or with right sided lesions in the lungs

**Clinical Findings** The patient may be attacked by malignant endocarditis during the course of some illness, such as pneumonia puerperal fever osteomyelitis, otitis media a cutaneous wound or a boil, or it may develop apparently spontaneously with or without a previous valvular lesion The patient is taken ill with malaise and a high swinging temperature the cause of which is uncertain The pulse is frequent and the temperature irregular The heart is usually dilated and varying cardiac murmurs are heard There is generally progressive anæmia and sweats or rigors ensue Embolic foci or hæmorrhages are indicated by pain in the region of the spleen by petechial spots or areas of redness in the skin, by hæmaturia cerebral symptoms or by diarrhœa A primary focus is usually discoverable such as osteomyelitis With the onset of endocarditis the patient becomes more gravely ill There may be drowsiness and mental apathy which with diarrhœa are suggestive of an enterica group infection

The blood shows a progressive leucocytosis and with counts over 15 000 per c mm the blood culture is usually positive There is also generally a hypochromic anæmia The urine Albuminuria hæmaturia and the causative organism may be present

**Course and Complications** The course varies with the response to the antibiotics The embolic manifestations mentioned above can be regarded as complications

**Differential Diagnosis** The diagnosis often presents difficulties especially when a case is seen for the first time during the illness and it is not known whether the cardiac murmur is of long standing Malignant endocarditis requires to be differentiated from such conditions as acute simple endocarditis subacute infective (bacterial) endocarditis enterica group infections miliary or acute tuberculosis septicæmia malaria abortus or Malta fever perinephric abscess subphrenic or hepatic

abscess meningitis malignant typhus or small pox The cardiac signs are of paramount value in the diagnosis

**Prognosis** This has been much improved by adequate treatment with antibiotics

**Treatment** The patient must be kept quietly at rest in bed Penicillin should be given The average dose is 500 000 units of crystalline penicillin every 6 hours In some cases as much as 8 million units every 24 hours are necessary to control the infection and lower the temperature If the frequent injections are not well tolerated 1 to 2 million units of a procaine preparation should be injected every 12 hours The treatment is usually required for 1 to 2 months Search should be made for a septic focus especially in the teeth If a satis factory response is not obtained in staphylococcal or streptococcal infections aureomycin should be given in addition to the penicillin in doses of mg 250 4 times daily by mouth for a month Alternatively erythromycin mg 500 can be given by mouth 4 times a day Strepto mycin combined with sulphadiazine should be given for H influenzæ infections The dosage is given on p 155

## Subacute Bacterial Endocarditis

### (*Endocarditis Lenta*)

**Definition** A progressive bacterial inflammation of certain parts of the endocardium which tends to run a prolonged course

**Etiology** The disease is caused by infection with the streptococcus viridans in over 90% of cases This is a streptococcus of the salivary or fæcal type *Predisposing causes* 1 Chronic rheumatic valvular disease 2 Congenital heart disease such as bicuspid aortic valves pulmonary stenosis ventricular septal defects and patent ductus arteriosus 3 Focal infection especially in the teeth or tonsils An attack may follow dental extractions or tonsillectomy or an infection of the upper respiratory tract

**Pathology** The aortic or mitral valve is usually affected and there may be no evidence of previous valvular trouble The vegetations are large and greenish in colour but ulceration of the valves is not common The vegetations spread to the walls of the heart chambers The myocardium is often spared

**Clinical Findings** The patient is usually a young adult male He notices gradually increasing weakness with dyspnœa on exertion loss of weight sweating and feverishness at night There may also be pains and vague swellings in the joints In some cases the initial symptom is pain in the spleen or pleurisy due to a pulmonary infarct in cases associated with congenital cardiac lesions

*On Examination* The complexion gradually becomes sallow (*cafe au lait* tinge) and the fingers may be clubbed Small painful red spots (Osler s nodes) may form and subsequently disappear These are embolic in origin They are seen on the palmar aspects of the tips of the fingers and toes Petechial hæmorrhages may occur especially around the neck and shoulders or under the nails (splinter hæmorrhages)

The temperature is usually about 99° to 100° F and runs an irregular course often being normal for long periods The pulse is frequent about 100 to 120 The heart at first often shows no abnormality later it dilates and a systolic murmur appears at the apex The appearance of an aortic diastolic murmur is of great significance indicating endocarditis The blood There is a progressive anæmia, with no leucocytosis or a moderate one of about 10 000 per c mm The blood culture is usually positive at some period of the disease The urine Minute bacterial emboli in the renal glomeruli result in hæmaturia from time to time Emboli *occur in other sites such as the spleen, the brain, the* retinæ, the large arteries of the limbs or the mesentery The cutaneous lesions mentioned above are probably embolic in nature An enlarged spleen can often be felt

**Differential Diagnosis** It is difficult to make a certain diagnosis in the early stages Diagnosis is established by the positive blood culture and by the appearance of cardiac murmurs and of embolic *phenomena*

**Course and Complications** The course is prolonged unless adequately treated the illness often lasting for 1 to 2 years Complications include the embolic processes described above

**Prognosis** This is grave, but has been much improved by the use of antibiotics

**Treatment** *Prophylactic* In the presence of congenital heart disease or chronic valvular disease a dental extraction or other operative *procedure should be covered with injections of penicillin for 24 hours* before and after the operation

*Curative* This is the same as described for acute bacterial endocarditis Relapses are not infrequent a few weeks after treatment is completed necessitating a second course of treatment This usually means a focus of infection remains The teeth should always be X rayed during the first course of treatment and if an apical abscess is present the offending tooth should be removed It may also be advisable to remove infected tonsils at the end of the first course of treatment

## Non Bacterial Endocarditis

Several varieties are described such as the rheumatic *which is the* most important the simple thrombotic which is not associated with rheumatism the verrucous which occurs in disseminated lupus erythematosus and the calcareous In verrucous endocarditis no Aschoff bodies are found in the heart muscle and the vegetations are *larger than* those found in rheumatic endocarditis and are spread over the *mural* endocardium

## Acute Rheumatic Carditis

**Definition** An acute infective inflammation of certain portions of the endocardium and myocardium, due to the rheumatic state which pursues a relatively benign course

**Etiology** *The endocarditis is associated with rheumatic* fever and chorea Less frequently it develops during an attack of tonsillitis or

scarlet fever A recurring endocarditis may appear in cases of long standing valvular lesions

**Pathology** The lesions probably result from the products of hæmolytic streptococci to which the tissues have become sensitised Vegetations of varying size form on the valves especially on the auricular surface of the mitral valve and the ventricular surface of the aortic valve and more rarely on the mural endocardium of the left auricle or ventricle Aschoff's nodules may be found in the myocardium There is always an associated carditis

**Clinical Findings** The patient is usually a young adult between *the ages of 10 and 20 who is suffering from rheumatic fever The onset* of acute endocarditis can only be detected by extremely careful and frequent clinical examinations Thus the pulse rate may gradually rise the temperature assume a slightly higher plane and changes appear in the cardiac signs The apex beat may move out slightly and a localised systolic murmur may be heard there owing to dilatation As the condition improves the apex beat may return to its normal position and the murmur disappear without there being any indication of permanent cardiac damage In other cases the systolic murmur may persist later a diastolic murmur appears indicating valvular damage Indications that the heart is definitely affected are the presence of a mid-diastolic an early diastolic or a presystolic murmur localised usually to a small area internal to the apex beat It is very difficult to hear the early or mid diastolic murmur unless the heart rate is slow If the heart recovers the early and mid diastolic murmurs may disappear and also the systolic murmur These early and mid diastolic murmurs are probably produced by the ventricle sucking the blood through an inflamed mitral valve An aortic diastolic murmur is less commonly heard Presystolic triple rhythm auricular fibrillation or flutter are other indications of carditis In some cases there may be a dry pericarditis which occurs early in the disease and is associated with pain restlessness vomiting and fever An electrocardiogram may show prolongation of the P-R interval indicating a degree of heart block which is restored to normal as the patient recovers

**Differential Diagnosis** The appearance of a soft diastolic murmur at the apex in the absence of mitral stenosis or the development of an aortic diastolic murmur is evidence of active carditis

**Course and Complications** The valvular lesion is often progressive Thus definite signs of mitral stenosis may appear in a few months Complications include pericarditis or pleurisy and congestive failure

**Prognosis** There is likely to be permanent valvular damage either slight and non progressive or severe with an increasing valvular defect Adherent pericardium may still further embarrass the heart s action

**Treatment** The patient must be kept slightly propped up in bed and not allowed to do anything for himself until the pulse temperature and respirations are normal He may then be taken off absolute ' rest but must lie quietly in bed for another month then he is gradually raised in bed with more pillows A careful watch must be kept upon the

size of the heart and the heart sounds during convalescence After 2 to 6 months in bed he is allowed to lie on a couch for graduated periods *if satisfactory progress is being made* The patient should not be allowed up until the sedimentation rate of the red cells is normal The salicylate treatment is as described for rheumatic fever (see p 601) Salicylates should be discontinued *if a cardiac depressant effect is* observed as shown by weakening of the pulse or dilatation of the heart Tab digitalis præp gr 1 t i d or larger doses according to the age of the patient and the response to the drug should be given if the pulse is rapid or there are signs of congestive failure If there is a heart block or a slow rhythm associated with premature systoles and coupled beats no digitalis should be given If there is any septic focus as in the tonsils intramuscular injections of penicillin should be given and the tonsils, if infected should be removed in the later stages of convalescence

### *Chronic Valvular Disease of the Heart*

**Definition** Inflammatory and degenerative changes in the valves of the heart

**Etiology** The *following varieties are described* 1 Congenital especially pulmonary stenosis 2 Inflammatory This may be rheumatic or a sequel of subacute bacterial endocarditis It may also result from influenza erysipelas pneumonia or septicæmia 3 Syphilitic The aortic valve and the anterior cusp of the mitral valve are liable to be affected 4 Arteriosclerotic and calcareous The aortic valve is involved

**Pathology** Deformity of the valve cusps or ring is likely to occur, but the mural endocardium remains practically unaffected

### Mitral Incompetence

**Definition** Reflux of blood from the left ventricle to the left auricle during the ventricular systole

**Etiology** The regurgitation may be due to dilatation or thickening of the valve ring or to alterations in the valve cusps or chordæ tendineæ preventing effective closure The valve ring The orifice may dilate in association with myocardial weakness and dilatation of the left ventricle in febrile or anæmic states or in association with enlargement of the left ventricle in aortic disease or increased blood pressure It may be thickened from organic changes associated with previous rheumatic or *other infections or with arteriosclerosis* The valve cusps and chordæ tendineæ may be deformed thickened and adherent as the result of inflammatory changes, and in some cases rupture may occur

**Pathology** In addition to the changes in the ring or valve cusps described above there is often in valvular cases a certain degree of narrowing of the valve orifice (stenosis) and some enlargement of the left ventricle, and later of the left auricle and finally of the right side of the heart

**Clinical Findings** The patient often gives a history of previous rheumatic infection He usually does not complain of any symptoms

unless the heart muscle itself begins to fail (failure of compensation) He will then notice a train of symptoms such as undue dyspnœa on exertion palpitations and later swelling of the ankles cough and expectoration

*On Examination* In a well marked case of fully compensated mitral regurgitation the only abnormal signs will be in the heart Inspection The apex beat may be displaced a little in the fifth and sixth spaces about 4 inches from the mid line Palpation The impulse is forcible and a systolic thrill is at times felt at the apex Percussion The area of cardiac dulness is increased a little downwards and to the left Auscultation Both sounds are heard at the apex and base At the apex there is a systolic murmur accompanying the first sound and in some cases apparently replacing it although graphically the first sound is always present It is maximum in late systole The murmur may be soft or loud and is conducted outwards towards the axilla in some cases being heard as far round as the angle of the scapula or even the spine The maximum intensity is over the apex The third heart sound is often heard after the second sound internal to the apex especially on inspiration A clicking opening snap may also be heard closer to the second sound than is the third heart sound and more widely distributed over the præcordium With failure of compensation other signs appear There is some cyanosis of the face and dyspnœa on slight exertion The apex of the heart may pass out further to the left and the rate increase Irregularity may be noted due to premature systoles or auricular fibrillation Further signs of venous engorgement may be found such as enlargement of the jugular veins in the neck dilatation of the tricuspid valve as indicated by a systolic murmur over the tricuspid valve area The liver may be enlarged and pulsating and rales may be heard at the bases of the lungs Œdema of varying degree may be seen in the ankles and legs and ascites may be present The urine is often diminished and contains albumin or blood

Differential Diagnosis Organic mitral incompetence is not diagnosed nearly so frequently now as it was a few years ago *The conditions under which a systolic murmur may be heard at the apex of the heart are as follows* —

1 *Physiological* In an apparently normal individual a soft systolic murmur may be heard at the apex of the heart This is due to a temporary dilatation of the valve ring and although it may produce no apparent disturbance of function it is not a normal condition

2 *Intracardiac* The murmur may be (*a*) *Hæmic* Associated with anæmia and dilatation of the valve ring and often heard better at the base than at the apex of the heart It is soft and often varies with position rest exercise and respiration (*b*) *Febrile* This is a soft and localised apical murmur which may occur during fevers without any evidence of dilatation of the heart but is probably due to dilatation of the valve ring (*c*) *Due to dilatation of the mitral valve ring* (*relative incompetence*) as in a dilated and hypertrophied heart This is a variety of mitral regurgitation (*d*) *Organic* Due to changes in the valve ring or segments as described above The murmur is blowing and

often loud and conducted towards the axilla The murmur is little affected by change of position, exercise or by respiration A mitral systolic thrill usually implies organic disease There is often enlargement of the left ventricle

3 *Exocardial* A cardio respiratory systolic apical murmur may be due to pleuro pericardial adhesions whereby the pressure of air in an adjacent portion of lung is affected by the heart beat The murmur is usually late systolic and is affected by the phases of respiration In overacting hearts the ventricular contraction causes the adjacent portion of lung to expand rapidly and suck in air, producing a cardio respiratory short and blowing murmur A systolic murmur may be heard in acute pericarditis, but more often there is a ' to and fro murmur

**Prognosis** In a simple case with a healthy myocardium, the heart being normal in size, the exercise tolerance good and there being no evidence of infection a full and active life may be expected for many years If there is aneurysmal dilatation of the left auricle or enlarge ment of the left ventricle heart failure is likely to ensue before the age of 50

**Treatment** No treatment is required unless there are indications of failure of compensation The patient must then be instructed to live well within his reserve of cardiac power and to avoid all strenuous exercise If decompensation occurs the treatment is as described on p 240 for the failing heart

## Mitral Stenosis

**Definition** Narrowing of the mitral valve

**Etiology** Mitral stenosis is usually inflammatory in origin being a sequel of acute rheumatic endocarditis in about 90% of cases but it also follows other infections such as scarlet fever or influenza Signs of stenosis do not usually show themselves before 2 years after an acute rheumatic attack Less frequently mitral stenosis is an athero matous lesion occurring in people over middle age. Fibro elastosis may occur in infancy with thickening of the endocardium due to the formation of fibrous and elastic tissue especially in the left side of the heart The mitral and aortic valves may be affected, and mitral stenosis may develop Sex Females predominate

**Pathology** The valve ring may be narrow, sclerotic and slit like (Corrigan s button hole stenosis) or the cusps may be adherent and the chordæ tendineæ shortened (funnel shaped stenosis) The former is more common in adults the latter in children Secondary changes appear in the heart and elsewhere such as dilatation and hypertrophy of the left auricle and right ventricle congestion of the lungs and liver etc

**Clinical Findings** In a typical case in which symptoms of failure of compensation are not marked the patient is often a young adult who gives a history of an infection particularly that of rheumatic fever some years before In many cases however no such history can be obtained The patient may notice no symptoms or he may complain of pal pita

tions dyspnœa on exertion or slight swelling of the ankles towards the evening

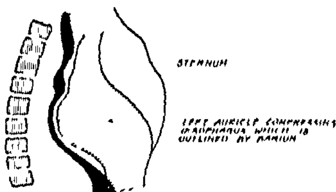
*On Examination* The mitral facies may be noted, in which the cheeks and lips have a high colour with slight cyanosis of the face and ears The cyanosis is probably due to vascular constriction The heart Inspection The apex beat is usually visible in its normal site but cardiac enlargement occurs with right ventricular hypertrophy Palpation The impulse is short forcible or slapping at the apex A presystolic apical thrill may be felt This is a rough vibrating sensation comparable with that experienced when the hand is placed on the back of a purring cat It can be timed by simultaneously feeling the carotid impulse Percussion The right border of the cardiac dulness may be a little out Auscultation The characteristic murmur in mitral stenosis is a coarse low pitched rumbling sound occurring in diastole The first sound at the apex is usually accentuated and the second sound rather weak The pulmonary second sound is accentuated The duration and intensity of the diastolic murmur depend upon the degree of stenosis and the rapidity and force of the heart beat In the early stages a short presystolic murmur is only heard after the patient has exercised as by sitting up and lying down 20 times the apex region being then auscultated with the patient lying on the left side Inhalation with amyl nitrite m 5 may also unmask the murmur The presystolic murmur is usually localised to a point just internal to the apex beat Later the murmur is more obvious extending through diastole The murmur does not begin immediately after the second sound The murmur is loud in early diastole it becomes fainter during mid diastole and louder again just before systole This presystolic crescendo is due to the auricular contraction and is not present when the auricles fibrillate Attention should also be paid to what is known as the mitral 'opening snap Normally the mitral valve opens silently In mitral stenosis as the valve opens a short clicking sound is heard directly after the second sound being higher in pitch than the second sound It is best heard in the third and fourth left spaces close to the sternum The diastolic murmur follows the opening snap

In early cases no diastolic murmur may be audible even after exercise etc Mitral stenosis may be suspected then by the first sound at the apex being loud and sharp In mitral regurgitation the first *sound is usually weak The pulmonary* second sound may also be accentuated or split

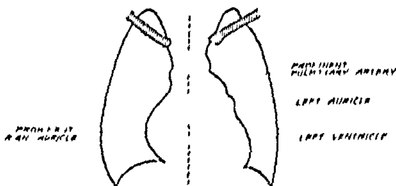
With fibrillation signs of failure of compensation are often present The pulse is usually of low systolic tension but the diastolic reading may be raised

*Radiographic Findings* A postero anterior X ray in a developed case shows prominence or dilatation of the pulmonary artery conus arteriosus and left auricular appendix The pulmonary artery becomes prominent owing to hypertrophy of the right ventricle The aorta is small The vascular shadows at the hilum are engorged In the right anterior oblique position in which the patient is rotated with the right

shoulder forwards through 45°, a shadow in the retrocardiac space due to the dilated left auricle may be seen. Calcification may be apparent in the mitral valve, in the centre of the heart shadow. Pulmonary haemosiderosis may cause a micro-nodular infiltration of the lungs. With a barium swallow the oesophagus may be seen curving round the dilated left auricle (see Fig. 15). The electrocardiogram is likely to show right axis deviation due to a vertical heart, with big or bifid P waves in




*(a) Right Anterior Oblique or Oblique I.* (The patient is turned so that the right shoulder rotates forward through 45 degrees.)




(b) P–A position

FIG. 15. DIAGRAM OF RADIOGRAPHIC APPEARANCES OF THE HEART IN MITRAL STENOSIS

leads I, II and $V_4$, $V_5$ and $V_6$. There may also be evidence of right ventricular hypertrophy and partial right bundle branch block.

**Differential Diagnosis.** Accurate timing of apical murmurs is essential. When this is done difficulty usually only occurs in the early stages, when there may be doubt between a short presystolic murmur heard after exercise and an accentuated or reduplicated first sound at the apex. Undoubtedly many cases have been diagnosed as early mitral stenosis, when the subsequent course has shown that no stenosis is present. With aortic incompetence a diastolic murmur (Austin Flint murmur, see p. 260) may be heard at the apex. In adherent pericardium a presystolic murmur may rarely be heard at the

apex although there is no mitral stenosis this is due to dilatation of the valve ring and is associated with a systolic apical murmur

**Course and Complications** Mitral stenosis usually pursues a prolonged course, and auricular fibrillation and heart failure do not necessarily ensue Complications include Embolism a clot may be detached from the left auricle and settle in the brain spleen or kidneys etc or from the right auricle and cause pulmonary embolus with hæmoptysis Congestion of the lungs hæmoptysis and bronchitis Premature systoles auricular fibrillation and heart failure Auricular fibrillation this is often of short duration at the onset but later it tends to be permanent Each attack of heart failure is more serious Laryngeal paralysis from pressure of the dilated left auricle on the left recurrent laryngeal nerve and pressure of the auricle on the left bronchus may cause pulmonary collapse Recurrent attacks of endocarditis may occur

**Prognosis** The presence of mitral stenosis usually means that the patient s life is limited both in activity and in duration It is a more serious lesion than mitral incompetence and death often occurs before the age of 40 It may cause sudden death usually from cerebral embolus

**Treatment** The patient must be instructed to live well within his reserve of cardiac power Failure of compensation and auricular fibrillation are treated as described on pp 228 240 Advances in thoracic surgery in the last few years and information on the pulmonary pressure and cardiac output gained by cardiac catheterisation have enabled some successful cases of mitral valvotomy to be performed The cases most suitable are those of pure mitral stenosis with a satisfactory myocardium but progressive lung symptoms and signs The pulmonary hypertension must not be too far advanced Embolism is also an indication for operation The mortality from the operation of valvotomy is about 10% Contraindications are active carditis associated aortic reflux considerable mitral incompetence gross enlargement of the heart or left auricle and auricular fibrillation or congestive failure of long standing

## Aortic Incompetence

**Definition** Reflux of blood through the aortic valve

**Etiology** Five types are described 1 Arteriosclerotic A degenerative lesion usually associated with syphilis 2 Endocarditic This is met with in young people especially as a complication or sequela of acute rheumatism 3 Traumatic A valve cusp may rupture from sudden strain if it is previously diseased by syphilis or subacute bacterial endocarditis 4 Congenital Two valve cusps may be fused so that they do not close properly 5 Relative incompetence Dilatation of the first part of the aorta and of the aortic ring may prevent closure of the cusps

**Pathology** The aortic valve cusps may be puckered and shrunken with associated stenosis or show vegetations and destructive lesions due to endocarditis There is usually hypertrophy of the left ventricle

the heart being enlarged (cor bovinum) There may be fibroid myocardial changes and atheroma of the aorta and coronary arteries

**Clinical Findings** The patient, who is usually an adult *male* may give a history of an attack of rheumatic fever some years ago or of having contracted syphilis earlier in his life He may complain of giddiness or faintness at times, or of headache, palpitations and dyspnœa on exertion In some cases præcordial pain is the *first symptom* noticed At times the onset is sudden with fainting or severe dyspnœa

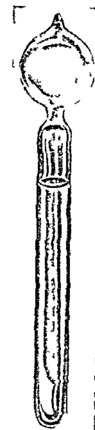


FIG 16 WATER HAMMER

*On Examination* Facial pallor is a characteristic feature although some authorities consider it is only a sign of bacterial endocarditis The patient may have an anxious expression, and pulsation of the carotid arteries may be noticeable The heart Inspection The cardiac impulse is forcible and diffuse, and the apex beat is in the sixth or seventh left space, external to the nipple line Pulsation may be seen in the supra sternal notch Palpation The impulse is forcible and usually displaced downwards and outwards There is generally no thrill Rarely a diastolic thrill is felt at the aortic base Percussion The area of cardiac dulness is increased downwards and to the left Auscultation Both sounds are usually heard at the apex There may be a systolic murmur at the apex due to mitral regurgitation An Austin Flint murmur may be heard at the apex This is a rumbling long diastolic murmur described by Flint in 1862 as blubbering which is thought to be due to the pressure of regurgitant blood in the aorta on the anterior cusp of the mitral valve At the aortic base or at times at the pulmonary base the second sound is replaced by a diastolic murmur or the second *sound may be heard accompanied by a murmur* The diastolic murmur is usually soft When heard at the aortic base it is usually conducted down to the xiphisternum and more rarely up into the neck When present over the pulmonary base it may be conducted downwards along the left border of the sternum towards the apex This is believed by some to indicate a lesion of the left posterior cusp of the aortic valve The first sound at the aortic area may be normal, or accompanied or replaced by a systolic murmur if there is roughening or stenosis of the aortic valve The rhythm is usually regular and the rate may be normal or increased The arteries are often thickened The pulse This is typically of the collapsing type and it is known as the water hammer or Corrigan s pulse The water hammer (*see Fig 16*) is a hermetically sealed tube exhausted of air and partly filled with water On inverting it the water strikes the far end of the tube with a sharp tap Sir Dominic Corrigan practised in Dublin and was physician to

The character of the pulse is best experienced if it is felt with the middle of the fingers rather than with the tips the patient s arm being first at a low level and then elevated In the latter position a short sharp tap is felt with systole and the characteristic collapsing sensation with each diastole The blood pressure The systolic pressure is high and the diastolic pressure low such as 170/60 mm Hg there being a high pulse pressure The blood pressure in the legs is higher than that in the arms this is due to a compensatory mechanism to maintain the cerebral circulation. On auscultation over the femoral artery a Duroziez murmur may be heard Duroziez, a Frenchman in 1861 said that the double intermittent crural murmur which had previously been described in aortic insufficiency was a constant sign of this lesion On listening over the femoral artery when the stetho scope is lightly applied a pistol shot sound is heard with each pulsation On pressing with the finger over the artery about ¾ inch above the stethoscope a systolic murmur is heard On pressing with the finger over the artery the same distance below the stethoscope there is a diastolic murmur Capillary pulsation (due chiefly to vaso-dilatation) On compressing the tip of the nail so as partially to blanch the nail bed pulsation may be seen at the junction of the white and red areas similarly by drawing the finger nail along the forehead and causing a red line to form capillary pulsation may be seen at the edges of the line Capillary pulsation may be seen inside the lips when they are compressed with a glass slide or in the retinal vessels with the aid of the opthalmo scope

**Differential Diagnosis** An aortic diastolic murmur may be very difficult to hear in the early stages absolute silence in the room being essential The patient should be examined sitting well forward erect and lying with the breath held in full inspiration and in full expiration both before and after exercise and auscultation should be practised along the right and left borders of the sternum In a well developed case the murmur is one of the easiest to detect There are then usually the concomitant signs of aortic reflux mentioned above

**Course and Complications** Aortic incompetence often pursues a prolonged course with little effect upon function so that for instance the patient can play a hard game of tennis Failure to maintain the cerebral circulation causes giddiness or faintness Recurrences of endocarditis will result in fever and possibly in embolism Extension of atheromatous changes may cause angina or aneurysm Heart failure with normal or abnormal rhythm results from myocardial weakness Aortic stenosis and mitral regurgitation place additional strain upon the myocardium but a combination of aortic regurgitation and mitral stenosis did not prevent a man from being a successful marathon runner Hemiplegia may result from cerebral hæmorrhage

**Prognosis** In traumatic cases sudden or rapid death is the rule With recurrent bacterial endocarditis death usually occurs in a few months to a year or so The most favourable outlook is in cases due to a previous endocarditis which is completely arrested With degenerative lesions coronary disease and myocardial failure usually eventually ensue

When heart failure occurs the prognosis is more unfavourable than is the case with mitral lesions In any case the possibility of sudden death must be remembered

**Treatment** The Wassermann reaction should be determined and if positive, a course of anti syphilitic treatment given as described on p 596 Anæmia may be helped by a mixture containing Ferri et ammon cit gr 15 to 20 sod bicarb gr 10 sp chlorof m 7 infus gent co rec ad fl oz 1 Fl oz 1 t d s p c For heart failure or auricular fibrillation digitalis should be administered as described on p 228 Pain is an indication for more complete rest

### Aortic Stenosis

**Definition** Narrowing of the orifice of the aortic valve

**Etiology** Aortic stenosis usually results from inflammatory changes following rheumatic endocarditis or in elderly people it may be caused by calcareous degeneration in the cusps This has probably developed on an old rheumatic infection Rarely if ever is it due to syphilis Congenital subaortic stenosis occasionally occurs or stenosis of the valve itself

**Pathology** The valve cusps are thickened shrunken and adherent They lose their mobility and the lumen of the valve orifice is narrowed Calcareous particles may be found in the cusps The valve cusps may be judged post mortem to be incompetent although no regurgitant murmur was detected during life The left ventricle is usually hypertrophied and mitral incompetence is often present In relative aortic stenosis the orifice is normal in size but the aorta beyond is dilated

**Clinical Findings** The patient is generally a male over middle age A history of rheumatic fever, and rarely of syphilis may be obtained He may complain of præcordial pain or distress on exertion of syncopal attacks due to exaggeration of the carotid sinus reflex or of symptoms due to some complication such as a retinal venous thrombosis disturbing vision

*On Examination* The patient often appears healthy The heart Inspection The cardiac impulse is forcible and the apex beat is seen in the fifth or sixth left space a little external to the nipple line Palpation A rough systolic thrill may be felt over the aortic base The apex beat is forcible Percussion The area of cardiac dulness is increased especially downwards and to the left Auscultation At the apex the first sound may be replaced or accompanied by a systolic murmur due to mitral regurgitation The second sound at the apex is weak Over the aortic base a rough systolic murmur is heard conducted upwards into the neck on the right side and also downwards over the sternum The murmur however becomes weaker before the point is reached at which the apical systolic murmur is picked up The aortic systolic murmur is loudest in mid systole The aortic second sound is usually weak and may be absent An aortic diastolic murmur may be heard indicative of aortic reflux (double aortic disease) The pulmonary second sound is often weak and may be practically inaudible if tricuspid regurgitation is also present so that it is difficult to hear a second sound

at any point over the heart The aortic systolic murmur may at times be heard at the back the maximum intensity being just to the left of the fourth thoracic vertebra The rate of cardiac beat is often slow such as 50 to 60 and the rhythm regular The arteries The radial or brachial arteries are often thickened The pulse This presents the slow small but sustained type of impulse the artery remaining filled between the beats A tracing shows the anacrotic type the dicrotic wave being absent or poorly marked The blood pressure The systolic pressure is not raised but the diastolic reading may be high such as 140/100

**Differential Diagnosis** Aortic stenosis is a rare disease and must not be diagnosed solely on the presence of an aortic systolic murmur This may only indicate roughening of one of the valve cusps atheroma of the aorta aneurysm overaction of the left ventricle or anæmia The diagnostic features are the weak aortic second sound the systolic murmur propagated to the neck and the small anacrotic pulse If the aortic second sound is absent there is probably extensive valvular disease

**Course and Complications** The lesion is usually slowly progressive Complications include bacterial endocarditis angina pectoris and congestive failure The latter is of grave significance

**Prognosis** Death usually occurs before the age of 50

**Treatment** The Wassermann reaction should be determined and if positive a course of anti syphilitic treatment should be given as described on p 596 In all cases strain and over exertion must be avoided Valvotomy is less successful than in mitral or pulmonary stenosis as there is a risk of producing aortic incompetence

## Tricuspid Incompetence

**Definition** Regurgitation of blood from the right ventricle to the right auricle

**Etiology** Tricuspid incompetence is usually associated with dilatation of the right ventricle and secondary to valvular lesions in the left side of the heart It may occur with pulmonary fibrosis chronic bronchitis and emphysema (cor pulmonale) Rheumatic endocarditis causing incompetence rarely affects the tricuspid valve A congenital variety may also be met with

**Clinical Findings** The onset may be comparatively sudden with acute right sided heart failure

*On Examination* The face is cyanosed Venous engorgement may be apparent in the neck the jugular veins being distended and pulsating with each ventricular systole The jugulars when emptied by the finger will be seen to fill from below The liver may be enlarged tender and on bimanual palpation systolic expansion may be felt The heart Palpation It is uncommon to feel a systolic thrill over the tricuspid area Percussion The heart is enlarged to the right Auscultation There is a soft systolic murmur with its maximum intensity over the fourth right costal cartilage and lower part of the sternum It may be conducted a little towards the right nipple or

heard at the back near the angle of the right scapula The pulmonary second sound is faint Other murmurs due to lesions of the mitral or aortic valves may be present There are usually rales at the bases of the lungs and ascites may be present

**Differential Diagnosis** The systolic murmur must be differentiated from that due to mitral incompetence The characteristic features of tricuspid regurgitation have been detailed above

**Course and Complications** Relative incompetence may disappear with adequate treatment

**Prognosis** This is serious, as the lesion is indicative of a severe degree of heart failure

**Treatment** Venesection and digitalisation are usually required

### Tricuspid Stenosis

**Definition** Narrowing of the orifice of the tricuspid valve

**Etiology** Tricuspid stenosis is a very rare lesion either resulting from previous rheumatic endocarditis or being congenital in origin

**Pathology** The narrowed tricuspid orifice is often associated with mitral stenosis

**Clinical Findings** The patient may be cyanosed and the skin of the face and neck pigmented Despite evidence of right heart failure the patient may show little distress The heart Palpation A presystolic thrill may be felt over the right side of the lower part of the sternum Percussion The heart is enlarged to the right Auscultation A diastolic murmur usually mid diastolic is heard with maximum intensity over the lower part of the sternum, conducted slightly upwards and to the right X ray examination shows considerable enlargement of the right auricle The electrocardiogram shows tall sharp P waves in lead II which are often wide if mitral stenosis is also present The liver It may be possible to detect presystolic (auricular systolic) pulsation The liver is usually enlarged firm and not tender (cardiac cirrhosis) There is frequently œdema of the ankles and ascites develops later

**Differential Diagnosis** As tricuspid stenosis is so rare it must be very carefully differentiated from mitral stenosis with which it is usually associated With the onset of auricular *fibrillation the diastolic* murmur usually disappears

**Treatment** This is as for auricular fibrillation or for right sided cardiac *failure (see pp 228, 237) Valvotomy has been occasionally* successful

### Pulmonary Regurgitation

**Definition** Regurgitation of blood through the pulmonary valve

**Etiology** Pulmonary regurgitation may occur as a complication of mitral stenosis due to increased pressure in the pulmonary circuit with dilatation of the pulmonary artery It may also be due to infective endocarditis grafted on a congenital pulmonary stenosis or more rarely it occurs as a congenital lesion combined with pulmonary stenosis It is a very rare valvular lesion

**Clinical Findings** The characteristic sign is a diminuendo soft diastolic murmur following the second sound with maximum intensity in the second and third left spaces near to the sternum It is conducted down the left border of the sternum The pulmonary diastolic murmur which may be heard in advanced cases of mitral stenosis is known as a Graham Steell murmur and is thought to be due to pulmonary regurgitation It is known as the murmur of pulmonary hypertension and when first developed is not usually constant

**Differential Diagnosis** It is very difficult to diagnose pulmonary regurgitation from early cases of aortic reflux in which the pulse is not collapsing and other signs of aortic disease are absent

### Pulmonary Stenosis

**Definition** Narrowing of the pulmonary valve

**Etiology** Pulmonary stenosis is usually a congenital lesion and is described on p 267 It may be due to compression of the pulmonary artery by a mediastinal tumour or an aortic aneurysm Rarely it is caused by bacterial endocarditis

## CONGENITAL DISEASE OF THE HEART

**Etiology** Congenital heart disease is usually caused by developmental errors more rarely by fœtal endocarditis The right side of the heart is generally affected probably owing to the higher pressure which obtains there during fœtal life It is more common in boys particularly in first born children There appears to be a relationship between the occurrence of congenital defects cardiac ocular and deaf mutism in the child and an attack of German measles in the mother during pregnancy especially during the first two months of pregnancy

**Pathology** Congenital lesions may be classified as —

1 *Abnormalities of Position* The heart may be situated external to the chest wall in the neck or in the abdomen (ectopia cordis) or it may be on the right side of the body (dextrocardia) with transposition of the aorta and pulmonary artery In the latter case the other viscera may be in their normal site or transposed

2 *Septal and Fœtal Passage Defects* (*a*) The interauricular or interventricular septum may be absent (cor triloculare) (*b*) The interauricular and interventricular septa may be absent (cor biloculare) (*c*) The interauricular septum may be imperfect or a patent foramen ovale may be present (*d*) The interventricular septum may be incomplete usually at the site of the pars membranacea near the upper end of the septum (*e*) The ductus arteriosus may remain patent Normally this closes by the eighth day after birth and if it remains patent it does so as a mechanism compensatory to other defects These include pulmonary stenosis and aortic stenosis the patent ductus allowing blood to pass to the pulmonary artery from the aorta in the former case and to the aorta from the pulmonary artery in the latter A patent interventricular septum is also often present

3 *Defects of the Main Vessels* (*a*) Pulmonary stenosis This m imply actual atresia of the pulmonary artery or narrowing of the art at the valve level or at the infundibulum (*b*) Aortic *stenosis* T occlusion may occur at the valve ring between this and the entra of the ductus arteriosus or at the point just below the entrance of ductus arteriosus (*coarctation of the aorta*, see p 269)

4 *Valvular Defects* (*a*) The pulmonary valve Pulmonary steno may be due to shrinkage of the valve cusps, or to narrowing of the val ring or a supernumerary cusp may be present often associated with patent foramen ovale or persistent ductus arteriosus Only two cus may be present or there may be a supernumerary one Pulmona regurgitation is uncommon (*b*) The tricuspid valve Congenit regurgitation or stenosis may occur (*c*) The aortic valve Two cus only may be present and the valve be incompetent, or there may be supernumerary cusp (*d*) The mitral valve This is rarely stenosed

5 *Defects of Conducting Tissue* Congenital heart block is usual due to a deficiency of the upper part of the interventricular septum t auriculo ventricular bundle ending in a band of fibrous tissue

6 *Combined Lesions* such as *Fallot s tetralogy* In this there stenosis or hypoplasia of the pulmonary artery a defect in the inte ventricular septum the aorta communicates with both ventricles an the right ventricle is hypertrophied The *Eisenmenger complex* diffe from Fallot s tetralogy inasmuch as the right ventricular conus an pulmonary valve are normal and the pulmonary artery is dilated

In the *Lutembacher complex* there is an interauricular septal defec combined with mitral stenosis It is generally believed that the latt lesion is acquired and not congenital in origin

**Clinical Findings** Cases of congenital heart disease may be classifie as cyanotic and acyanotic The cyanotic are blue when at rest owin to a venous arterial right to left shunt Some cases show a border lin cyanosis which may slowly increase owing to changes in intracardia pressure or to heart failure *The cyanotic group* includes Fallot tetralogy, the Eisenmenger complex pulmonary valvular stenosis wit patent foramen ovale, pulmonary atresia tricuspid atresia and trans position of the aorta and pulmonary artery *The acyanotic grou* embraces patent ductus arteriosus auricular septal defect ventricula septal defect pulmonary valvular defect with closed septum an coarctation of the aorta

Campbell also classifies cases of congenital heart disease int *oligœmic* with diminished blood flow to the lungs and hypertranslucent lung fields as seen on X ray examination and *pleonœmic* with increased blood flow to the lungs and dense lung fields as seen radiographically Here there is a left to right shunt The importance of this subdivision lies in the fact that the blood flow to the lungs can be increased by the Blalock Taussig operation (see p 269) The table on page 267 shows Campbell s classification

In a typical case in which there is deficient aeration the condition is noted at or shortly after birth The baby is cyanosed and blue (*morbus*

## Patent Ductus Arteriosus

Frequently there are no symptoms the patient is not cyanosed a the fingers are not clubbed The heart Pulsation may be seen in t second and third left spaces, near the sternum due to enlargement the pulmonary artery A systolic thrill may be felt at the site of t pulsation and a diastolic shock may be present Auscultation murmur may be heard which is harsh or blowing and begins just aft the onset of the first sound It fades away towards mid diastole a becomes loud again with each systole (water wheel or machine murmur) The murmur is maximal at the second or third left spa near the sternum, and it may be conducted towards the left clavicle heard at the back in the left interscapular region It is due to blo flowing from the aorta to the pulmonary artery The pulmona second sound is accentuated At the apex the second sound may l reduplicated The pulse may show an increased pulse pressure X ra examination often shows dilatation and pulsation of the pulmonar artery and pulsating hilar vessels The electrocardiogram is usual normal Angiocardiography may show the pulmonary artery fillin from the right ventricle first and then from the aorta Cardia catheterisation usually shows blood from the right auricle and ventric with a normal oxygen content (70% saturated) whereas blood fro the pulmonary artery is over 80% saturated Death usually occu before the age of 40 either from bacterial endocarditis or congestiv failure Paradoxical embolus may occur, a clot passing from the le auricle or from the mitral or aortic valve to the lung Surgical treatmen consists in ligation or excision of the ductus under penicillin cove This should prevent bacterial endocarditis developing If it is presen it should be eradicated by penicillin before operation The mortalit rate from the operation is about 3% in skilled hands The operatio must only be performed if the patent ductus is the only lesion present

## Patent Interventricular Septum

The heart Auscultation A loud or harsh murmur (*bruit de Roger*) may be heard which starts early in systole and extends into diastole It may be audible in the mid sternal line or along the left sterna border near the third costal cartilage or lower down near the xiphi sternum It is not conducted to the neck or to the axilla The pulmonary second sound is audible Many cases are symptomless in early life The lesion may be associated with congenital heart block or with Fallot s tetralogy Bacterial endocarditis may develop usually on the right side

## Patent Foramen Ovale

With a large atrial septal defect the blood passes from the left auricle to the right auricle There is enlargement of the right side of the heart and of the pulmonary artery The hilar branches of the pulmonary artery may reach aneurysmal size A systolic murmur is heard over the middle or upper part of the sternum on the left side and the pulmonary second sound is usually accentuated and split or

pulmonary incompetence may occur Right bundle branch block is found in about 95% of cases Cardiac catheterisation will determine whether a shunt is taking place between the auricles as in about 25% of normal people the foramen is patent although functionless Patent foramen ovale may be associated with mitral stenosis (the Lutembacher complex) or with arachnodactyly (Marfan's syndrome) In the latter the fingers and toes are elongated the face is thin the palate has a high arch the chest is pigeon shaped and the lenses are dislocated The atrial septal defect often causes no symptoms until middle life when heart failure or more rarely bacterial endocarditis may supervene In countries in which schistosomiasis is endemic the radiological appearances of the heart in bilharzial heart disease may be indistinguishable from those of atrial septal defect

### Coarctation of the Aorta

Two types are described 1 Adult The constriction is at the site of the ductus arteriosus 2 Infantile There is narrowing of the isthmus between the left subclavian artery and the ductus arteriosus The patient usually complains of no symptoms but intermittent claudication may occur

Signs of hypertensive heart disease are usually present The characteristic features of coarctation are The blood pressure is raised in the arms and low in the legs Animal experiments indicate that the hypertension in the arms is of renal origin The systolic brachial pressure may be 150 to 160 mm Hg in a child of 10 years and often no pulsation can be felt in the femoral arteries Enlarged and tortuous arteries may be seen or felt in the chest, especially in the intercapsular region or in the neck and arms X ray examination may reveal notching of the ribs due to the enlarged intercostal arteries (Roesler s sign) The ascending aorta dilates and the aortic knob is small Angiocardiography will reveal the aortic constriction Death may result from congestive failure rupture of the heart or aorta cerebral hæmorrhage or bacterial endocarditis Death usually occurs before the age of 40 Crafoord s operation of surgical repair has proved successful in many cases the operation mortality rate being about 10 to 16%

### Fallot s Tetralogy

This is a cause of cyanosis polycythæmia and clubbing of the fingers in adult life A systolic thrill and murmur are usually present near the sternum in the second and third left spaces The X ray findings are typical the heart having a *cœur en sabot* appearance the aorta is displaced to the right the right ventricle is hypertrophied and there is a concavity where the shadow of the pulmonary artery should be seen Angiocardiography which shows simultaneous filling of the aorta and pulmonary artery and cardiac catheterisation which confirms the presence of pulmonary stenosis are helpful in the diagnosis of doubtful cases The Blalock Taussig operation consists in anastomosing one or other subclavian artery with the homolateral branch of the pulmonary

artery The operation mortality rate is about 10% Pulmonary valvotomy is an alternative operation

### Tricuspid Atresia

In this condition there is intense cyanosis due to a right to left atrial shunt the left *ventricle is much enlarged* The *blood supply* of the lungs is reduced, and is maintained through a patent interventricular septum patent ductus or a bronchial collateral circulation The Blalock Taussig *operation is of value*

## ANEURYSM

**Definition** *An abnormal dilatation of an artery resulting from* changes in its walls

*Varieties* Aneurysms may be subdivided into 1 True aneurysms *The wall contains one or more of the arterial coats They may be* (*a*) Fusiform or dilatation The aortic arch is especially liable to be thus affected (*b*) Saccular A localised swelling may form on large or *small arteries* (*c*) *Dissecting A new channel is formed in the media* through which the blood flows for a variable distance before regaining its normal path (*d*) Cirsoid A small artery and its branches are *involved in a fusiform dilatation*

2 Arterio venous aneurysms These may be (*a*) An aneurysmal varix There is direct communication between an artery and a vein (*b*) *A varicose aneurysm The artery and vein communicate through a* sac

3 False aneurysms A swelling containing blood communicates *with an artery, but the walls of the swelling are not formed* by the arterial coats

The fusiform sacculated and dissecting aneurysms are of medical *importance the other varieties are chiefly of surgical interest* Small saccular aneurysms (" berry aneurysms) may occur in the cerebral or retinal arteries or elsewhere in the body They are of clinical import*ance when occurring in the brain* (*see p 314*)

### Aortic Aneurysm

**Etiology** Syphilis is the most important cause and owing to the more efficient early treatment of syphilis aneurysms are much more rare than they were formerly The fusiform type however, may be associated with arteriosclerosis and not with syphilis A fusiform aneurysm may also develop in association with coarctation of the aorta Cystic necrosis of the aorta is also a cause of dissecting aneurysm which may subsequently rupture Mycotic aneurysms occur in association with bacterial endocarditis and septicæmia, emboli being carried to the vasa vasorum of the arterial wall Such mycotic aneurysms are usually multiple *Predisposing causes* 1 Age Usually over 50 2 Sex Males especially 3 Occupation Involving strain

**Pathology** Syphilis causes a mesaortitis the elastic and muscular fibres degenerate and the wall of the aorta yields to the pressure of its contents The sac of the aneurysm is formed internally *of layers of*

blood clot outside which are the remains of the middle coat the outer coat, fibrous tissue and perhaps surrounding structures to which it becomes adherent, such as the sternum, vertebræ or trachea Calcareous plaques may be seen on its inner surface In the adjacent parts of the aorta there are usually found the scars of syphilitic aortitis In cystic necrosis of the aorta there is degeneration of muscular and elastic tissue in the media, with the formation of small cystic areas containing mucoid material About 75% of aortic aneurysms are thoracic involving the arch and about 10% are abdominal The clinical findings of aortic aneurysms will be considered under separate headings according to the portion of the aorta involved

### Aneurysm of the Thoracic Aorta

Broadbent taught that an aneurysm of the ascending part of the arch is one of physical signs whereas aneurysms of the transverse and descending parts of the aortic arch give rise chiefly to symptoms due to pressure effects

### Aneurysm of the Sinuses of Valsalva

**Clinical Findings** The patient may be a comparatively young man between 30 and 40 years of age He may complain of faintness præcordial distress headache or of pain resembling that of angina

*On Examination* Generally no signs of aneurysm are detected but there is evidence of aortic reflux In some instances owing to pressure of the aneurysm on the inferior vena cava there is œdema of the legs ascites and enlargement of veins in the abdominal wall Sudden death may occur from rupture into the pericardium In other cases the rupture may be more gradual and of a dissecting character

### Fusiform Dilatation of the Aortic Arch

**Clinical Findings** The patient is usually an adult male past middle age He may have no symptoms or he may notice a little difficulty in breathing or in swallowing In other cases the symptoms are characteristic of aortic reflux

*On Examination* Inspection The patient must be viewed in a good light both from the side and in front Pulsation may be seen just above the manubrium sterni or in the first or second right intercostal space close to the sternum or the manubrium sterni itself may pulsate Palpation The impulse may be felt at these sites The cardiac apex is usually forcible and it is often displaced a little downwards and outwards A systolic thrill may be present over the aortic base with a diastolic shock Percussion An area of dulness may be detected over the manubrium sterni Auscultation The aortic second sound may be clear and ringing or an aortic diastolic murmur may be heard There may also be an aortic systolic murmur The arteries These are usually thickened Absence of the brachial and radial pulses on both sides has been recorded probably owing to obstruction of the orifices of the innominate and left common carotid and subclavian arteries The blood The Wassermann reaction is not necessarily

positive X ray examination Screening in the postero anterior and oblique positions will reveal the dilatation of the aorta It should be remembered that pulsation can often be felt in the suprasternal notch without any dilatation of the aorta being present usually when the aorta is uncoiled In all doubtful cases of aortic aneurysm angiography is likely to be of value

## Saccular Aneurysm of the Ascending Arch

**Clinical Findings** The patient is usually an adult male past middle age He may give a history of syphilis years ago The symptoms are very variable Thus there may be no complaint of ill health or the patient may notice pain in the region of the sternum on the right side of the chest or in the back between the shoulders He may complain of swelling of the face, neck, arms or hands or of a feeling of engorgement of the face and neck particularly on stooping There may be dyspnœa on exertion or paroxysms of coughing Slight stridor may have been noted when the patient is asleep In some cases a throbbing is felt in the aneurysm or hæmoptysis may be the first symptom

*On Examination* *Inspection* *The face and neck may be high* coloured with injected conjunctivæ and one or both arms may be swollen Enlarged veins may be seen on the front of the chest When the patient stoops down there is often considerable congestion of the face and neck In rare cases when there is interference with the blood supply to one arm as in aneurysm of the innominate artery unilateral clubbing of the fingers may be present Slight pulsation may be detected in the second or third right space near the sternum or there may be a definite pulsating swelling the size of a golf ball or larger Palpation Pulsation may be felt in the sites mentioned above and with an aneurysm the pulsation may be expansile A systolic thrill is rarely felt The apex beat is usually forcible and displaced a little downwards and outwards Percussion Dulness may extend outwards from the manubrium sterni to the right in the first to third spaces for 1 or 2 inches Auscultation The aortic second sound is accentuated and a systolic murmur may be heard over the pulsating area The pulse The radial pulses may be unequal in force or in time *but this only* occurs in about 4% of aortic aneurysms A difference of blood pressure of over 20 mm Hg in the two arms is also of diagnostic significance *The lungs* *Pressure on the right eparterial bronchus* may result in slight dulness and weak air entry over the apex of the right lung The trachea Pressure here may cause dyspnœa stridor (the leopard growl) or a harsh cough (gander cough) The right recurrent laryngeal nerve This is rarely involved pressure produces first abductor paralysis and later a complete paralysis In the latter stage the cough is described as bovine lacking any explosive character The blood The Wassermann reaction is positive in a high percentage of cases

## Saccular Aneurysm of the Transverse and Descending Arch

**Clinical Findings** The patient is *likely to complain of pressure* symptoms He may notice shortness of breath either on slight exertion

or when at rest cough expectoration or difficulty in swallowing solid food There may be pain under the sternum in the back between the scapulæ round the chest or down the arms There may be swelling of the face the neck or the left arm The dyspnœa may be relieved if the patient stoops forward as this tends to increase the antero posterior diameter of the chest

*On Examination* Inspection Pulsation may be seen over the manubrium sterni or in the left interscapular region Palpation Pulsation or a thrill may be felt over the manubrium Percussion The heart may be enlarged downwards and to the left dulness may be present over the manubrium Auscultation The aortic second sound may be accentuated *Further signs of aneurysm* A tracheal tug may be felt if the cricoid cartilage is elevated with the forefingers while the patient lifts up his head The larynx may not move upwards with deglutition The air entry may be weak over the upper or lower lobe of the left lung and signs of bronchitis or of bronchiectasis may be found in the left lung More rarely signs of distention of the left lung are noted the note being hyperresonant and the breath sounds weak Pressure on the left recurrent laryngeal nerve may cause laryngeal paralysis The left pupil may be dilated from stimulation or con stricted from paralysis of the sympathetic There may be flushing or sweating limited to one side of the face corresponding with the side of the aneurysm Slowing of the heart may result from stimulation of the vagus Pressure on the phrenic nerve may cause hiccough or paralysis of the left half of the diaphragm Pain in the chest or left arm may be due to pressure on the intercostal nerves or the brachial plexus The left pulse may be weaker than the right if the aneurysm is situated between the innominate and the left subclavian arteries and the blood pressure lower in the left arm than in the right Lowering of blood pressure in the left carotid artery may also cause dilatation of the left pupil due to relaxation of the vessels in the left iris. Pressure on the thoracic duct may result in chylothorax and œsophageal pressure may cause dysphagia

### Aneurysm of the Descending Thoracic Aorta

**Clinical Findings** The patient may complain of a gnawing pain in the back in the thoracic region radiating around the chest or spreading to the lumbar area There may be pains in the legs or weakness or later paralysis Dysphagia cough and expectoration are also some times present

*On Examination* Inspection The back should be examined in a good light with the patient sitting up It should be viewed from side to side when a pulsating area may be seen usually low down to the left of the thoracic vertebræ Palpation Pulsation or a thrill may be felt in the left interscapular region or just below this area Percussion The note may be impaired over this area Auscultation A systolic murmur may be heard in the left interscapular region The arteries The radial pulses are equal but the femoral pulses may be weak or absent The lungs There may be signs of bronchitis or of bronchi

ectasis in the left lung or of a left pleural effusion In more advanced cases erosion of the bodies of the vertebræ results in kyphosis or compression paraplegia, with sensory and motor disturbances in the legs

**Prognosis** A thoracic aneurysm is a very serious disease, and recovery is not to be expected Death usually occurs between 4 and 15 years from the time of diagnosis

**Treatment** *Prophylactic* Syphilis should be treated adequately and early

*Curative* The anti syphilitic treatment described on p 596 should be given, but it is advisable to keep the patient in bed for at least 3 months after the diagnosis is made This will aid clotting by diminishing the circulation rate If there is venous engorgement venesection of 10 to 20 oz from the arm often affords much relief, and can be repeated as required The diet should be moderate A regular daily action of the bowels should be secured with the help of salts senna or cascara sagrada The pain is generally relieved by the iodides but if very severe Nepenthe m 10 to 15 may be required or tab pethidin hydro chlor mg 50 by mouth When the patient is allowed up all violent exercise or strain must be avoided

## Aneurysm of the Abdominal Aorta

Saccular, fusiform or dissecting aneurysms may develop A saccular aneurysm near the origin of the cœliac axis artery is perhaps the most common variety

**Clinical Findings** The patient complains of abdominal or lumbar pain It is a continuous type of pain and may be intensified by lying down

*On Examination* Inspection With a large aneurysm a pulsating swelling may be seen in the epigastrium or at times in the back near the lumbar vertebræ Palpation A pulsating swelling is felt in the abdomen This is definitely expansile The femoral pulses may be obliterated In some cases an aneurysm may form on a renal artery with the appearance of a large pulsating swelling in the loin

**Differential Diagnosis** A pulsating abdominal aorta is frequently noted in thin patients especially in women It does not usually cause pain although its throbbing may produce discomfort The swelling is not expansile the femoral pulses are normal and there are no pressure symptoms such as may result with an abdominal aneurysm from erosion of the vertebræ An abdominal tumour may receive trans mitted pulsation from the aorta If the patient is examined in the knee elbow position, the pulsation usually disappears as the tumour drops forward

**Treatment** This is as for thoracic aneurysm

## Dissecting Aneurysm

**Pathology** A dissecting aneurysm may form in any part of the aorta It is usually secondary to cystic necrosis of the media The

intima ruptures and blood tears up a path between the coats of the aorta Rupture of the dissecting aneurysm is usually a terminal event

**Clinical Findings** This is not an uncommon condition The patient is usually a male and often apparently in good health In other cases he is known to be suffering from hypertension In women about 50% of cases are associated with pregnancy The patient may be suddenly seized with agonising pain either in the chest between the scapulæ at the back of the neck in the loins or in the abdomen The pain may radiate to the head arms pelvis or legs In other cases there is no pain the onset being sudden with loss of consciousness and neurological signs may be found in the limbs The pulse is often slow and feeble according to the degree of shock and collapse In some cases hypertension persists in others there is extreme dyspnœa Swelling of the neck may be noted when the rupture occurs in the region of the aortic arch Extensive bruising appearing in a few hours in the skin of the neck chest or abdomen is very suggestive The electrocardiogram shows no evidence of coronary infarction

**Course and Complications** If rupture has not occurred the patient may recover from the initial attack only to have further attacks one of which proves fatal In some cases however the patient dies from another disease and the dissection is discovered at autopsy

**Differential Diagnosis** Such conditions as coronary occlusion an acute abdominal emergency a cerebral hæmorrhage or acute lumbago may be diagnosed In one of my cases the pain came on suddenly in the left lumbar region as the patient stretched up in bed to switch on the light Few cases are diagnosed during life

**Treatment** Morphine sulph gr $\frac{1}{4}$ or Omnopon gr $\frac{1}{3}$ should be injected and repeated as necessary to dull the pain

## Acute Polyarteritis Nodosa

### (*Periarteritis Nodosa*)

**Definition** A condition characterised by a panarteritis of the medium sized and small arteries often with the formation of small aneurysms

**Etiology** Acute polyarteritis nodosa is probably a manifestation of hypersensitivity to various antigens especially to bacteria Sulphonamides organic arsenical preparations iodine thiourea and desoxycorticosterone acetate have all been reported as sensitising agents

**Pathology** There is a necrotising panarteritis with fibrinoid necrosis affecting initially the media and subintima periarteritis occurring as a secondary process Healing occurs by granulation scarring and occasional aneurysm formation The aneurysms may rupture Sections of vessels are affected and the vascular changes may occur only in the vessels of one system or of one or two isolated organs The arteries affected may supply the heart kidneys mesentery liver stomach intestines spleen diaphragm brain lungs muscles or subcutaneous tissues

**Clinical Findings** The patient is usually a male between the ages of 30 and 40 but he may be a young child The onset of symptoms is

generally sudden, with fever, weakness, muscular or joint pains vomiting and diarrhœa In other cases there is a sudden onset with coma and convulsions or the illness may be characterised by severe abdominal or cardiac pain, hæmoptysis or hæmaturia Bronchial asthma may be a prominent symptom Urticaria and purpura may also occur Asymmetrical peripheral neuritis a mononeuritis multiplex, involving one nerve after another, may be the most prominent symptom although symmetrical peripheral neuritis is more common

*On Examination* Small nodules may sometimes be felt on the subcutaneous arteries of the chest or abdomen The urine may contain blood or albumin The blood pressure is raised in about 65% of cases The temperature is usually raised and the blood may show a leucocytosis of about 30 000 per c mm Subcutaneous nodules or eosinophilia are only found in about 20% of cases

**Differential Diagnosis** The nodules may suggest somatic tæniasis The fever and abdominal symptoms may resemble those occurring in enteric fever or in miliary tuberculosis The nervous symptoms are liable to be mistaken for meningitis or a cerebral hæmorrhage Acute renal pain may suggest a renal calculus or perinephric abscess The hæmaturia suggests acute nephritis The dyspnœa with eosinophilia may be mistaken for asthma or, if the X rays show transitory pulmonary infiltration the condition closely resembles Loeffler s syndrome It is rare for a correct diagnosis to be made before autopsy unless the nodules are felt attached to the subcutaneous arteries

**Course and Complications** The disease usually pursues a progressive course lasting 3 to 4 months but cerebral or perirenal hæmorrhage may cause sudden death

**Prognosis** The disease shows a mortality rate of about 50%

**Treatment** Penicillin, salicylates and cortisone have all been recommended Cortisone or ACTH is chiefly of value in the acute cases In hypertensive conditions it must be used with special caution

## Temporal Arteritis

*(Cranial Arteritis)*

This condition resembles in many respects polyarteritis nodosa Although it was originally stated that the changes in temporal arteritis occur primarily in the adventitia, it is now recognised that the media is frequently involved Temporal arteritis has been described in a case of polyarteritis nodosa The changes are not limited to the temporal arteries but may be seen in the occipital arteries and other branches of the common carotid, in the aorta and in the arteries supplying the viscera and the limbs

**Clinical Findings** Temporal arteritis affects adults after the age of 55 years The first symptom may be tenderness on wearing a hat There is often a prodromal stage of ill health with loss of weight malaise night sweats and low grade fever Later the headache appears in the temporal or occipital region it is severe and usually only relieved by morphine It is not uncommon for the patient to complain first of ocular symptoms, usually blurring of vision which may rapidly lead to

blindness in one or both eyes Pericarditis has been described as a complication The affected temporal artery stands out like a sinuous cord it is tender and nodules may be felt in its thickened wall The skin over it is red

**Treatment** Periarterial injection of 2 ml of 1% procaine may give complete relief of headache The treatment can be repeated if the headache recurs Removal of a section of the affected artery may result in the rapid relief of pain

## Arteriosclerosis

**Definition** Local or diffuse hardening of arteries due to inflammatory or degenerative changes in their coats

**Varieties** From the clinical standpoint arteriosclerosis may be divided into two main classes atherosclerosis which affects the aorta and its main branches and peripheral arteriosclerosis

### Atherosclerosis

**Etiology** Atherosclerosis is considered to be due to a defect in cholesterol metabolism *Predisposing causes* 1 Age The incidence increases with age 2 Sex Males predominate but after the menopause the incidence is about equal 3 High blood pressure 4 Hypercholesterolæmia 5 A hereditary diathesis 6 Overeating and overdrinking 7 Obesity

**Pathology** The early lesion consists of yellow streaks in the aorta composed of cholesterol phospholipids and protein It is situated in the intima Later the atheromatous plaque forms The aorta and large arteries are chiefly affected also the coronary and cerebral vessels and those supplying the lower limbs rather than the arteries of the arms Fatty degeneration may lead to an atheromatous ulcer and calcification may ensue

### Peripheral Arteriosclerosis

There are two main types medial sclerosis or Monckeberg s sclerosis and the diffuse hyperplastic sclerosis of Gull and Sutton

### Medial Sclerosis

**Pathology** Fatty degeneration and calcification occur in the media with the formation of pipe stem vessels The intima is secondarily thickened It commonly affects the arteries in old age and in association with diabetes mellitus Monckeberg said that the brachial arteries are seldom involved Despite this the common variety of arteriosclerosis characterised by thickening and tortuosity of the brachial arteries does not appear to fall into any of these categories but it is usually held to be a variety of medial sclerosis The blood pressure is not necessarily raised

### Diffuse Hyperplastic Sclerosis

**Pathology** The small arteries, arterioles and capillaries are chiefly affected and there is often some fibrosclerosis Hyaline degeneration occurs in the intima of the arterioles especially in the kidneys spleen

brain, pancreas, liver and suprarenals The parent arteries show intimal thickening from proliferation of the subintima In the capillaries the endothelium swells and degenerates Fibroid myocarditis and chronic nephritis are often present in adults and renal disease in children (see also p 476)

### Endarteritis Obliterans

Narrowing of the smaller arteries in the extremities occurs due to proliferation of endothelial cells This may result in ulceration or gangrene in the digits The cause is unknown it is met with in syphilis at times in tuberculosis, in rickettsial infections after injuries and in frost bite The arterial circulation in the limbs is normal and the pedal pulses are palpable when the toes are affected The arms are affected more often than the legs

**Clinical Findings** The patient is usually a male past middle age The onset of the disease is insidious and nothing abnormal may be noticed beyond a slight pallor *in some cases until the arterial changes* produce symptoms definitely located to some portion of the body In other cases when there is a diffuse thickening taking place in the various arteries of the body the patient may complain that he is be coming prematurely old and unfit for the activities to which he has been accustomed The effects produced by arteriosclerosis vary with the type of pathological change present but clinically they are best considered regionally The blood pressure is usually raised and the heart hypertrophied but in senile arteriosclerosis this is often not the case and it is not necessarily so in atherosclerosis The condition of the arteries may be judged clinically by palpating those which are superficial, such as the radial brachial facial temporal femoral and dorsalis pedis The radial artery may be firm like a whipcord in diffuse hyperplastic sclerosis or calcified nodules may be felt in atheroma or it may be hard like a pipe stem in the medial type The radial and brachial arteries are sometimes tortuous and this may occur without calcification Tortuosity of the temporal arteries does not *necessarily imply arteriosclerosis Ophthalmoscopic examination* may show arteriosclerotic changes in the retinal vessels

*Cerebral Symptoms* There may be mental deterioration lack of concentration and insomnia or attacks of giddiness temporary loss of consciousness paresis aphasia or epileptiform convulsions may occur Arteriosclerotic Parkinsonism is also described characterised by rigidity affecting especially the trunk and lower limbs a mask like expression and bradykinesis Transitory blindness due to spasm of the vessels may ensue Cerebral thrombosis or hæmorrhage are more serious complications

*Cardiac Symptoms* Atherosclerosis of the aorta or coronary arteries may lead to aneurysm of the aorta aortic regurgitation myo cardial degeneration cardiac hypertrophy angina pectoris coronary thrombosis or aneurysm of the heart followed by rupture

*Abdominal Symptoms* Attacks of abdominal pain constipation or mesenteric thrombosis may ensue

*Renal and Vesical Symptoms* Arteriosclerotic kidneys may result in chronic nephritis usually without œdema Hæmaturia may result from arteriosclerosis of vessels in the bladder

*Peripheral Symptoms* Involvement of the arterial supply of the extremities especially the legs results in intermittent claudication The patient finds he cannot walk more than a few yards without severe pain in the legs which ceases as soon as he rests In more advanced cases there may be pain and cramps apart from exercise and gangrene may slowly set in In the diffuse hyperplastic sclerosis the clinical picture is that of a systolic blood pressure of 180 mm Hg or more with secondary cardiac hypertrophy (hypertensive heart disease) This is known clinically as hyperpiesia or essential hypertension

**Differential Diagnosis** Arteriosclerosis is usually easily diagnosed by palpating the peripheral vessels In the medial form an X ray film may show the calcification Intermittent claudication due to arterio sclerosis must be differentiated from thrombo angiitis obliterans The characteristic features of the latter disease are that it occurs in younger men usually Hebrews and migrating phlebitis is also present in the legs

**Course and Complications** The arterial changes are progressive and such complications as hæmorrhage gangrene or muscular degeneration are liable to occur from interference with the blood supply to the tissues Bronchitis may develop

**Prognosis** The outlook is more unfavourable when calcification occurs in the arteries of a young man Myocardial degeneration aneurysm and renal inefficiency all increase the severity of the condition

**Treatment** This is in the main that of high blood pressure (see p 282)

For intermittent claudication the patient should be given nicotinic acid by mouth in doses sufficient to produce flushing beginning with mg 25 or 50 t i d This may afford great relief Alternatively Hydergine may be administered the patient taking 3 to 8 tablets (0 25 mg of the active ingredients in each) daily sublingually and 1 ml being injected subcutaneously once or twice a week With improvement the dose may be reduced to 1 to 3 tablets a day In addition hot baths massage and passive vascular exercises (see p 286) are of value

## Pulmonary Arteriosclerosis

(*Pulmonary Atheroma Ayerza s Disease Cardiacos Negros*)

There are two main groups of cases 1 Due to primary changes in the pulmonary artery and its branches 2 The changes in the pulmonary artery are secondary to changes in the lungs and heart such as chronic bronchitis emphysema and mitral stenosis The primary form may be due to a syphilitic infection of the pulmonary artery but there is a non inflammatory sclerosis occurring in younger people the cause of which is unknown Ayerza s case was secondary to long standing disease of the lungs

The clinical picture resembles that described under Pulmonary Heart Disease

### Phlebosclerosis

Hardening of the veins may occur in association with varicose veins in thrombo angiitis obliterans in arterio capillary fibrosis, and in diabetes mellitus The pulmonary veins may be affected in mitral stenosis

### Thrombosis of the Axillary Vein

This may occur in healthy young adults as the result of a sudden strain made with the arm and hand rarely it develops spontaneously It may be due to pressure of the costocoracoid ligament and subclavius muscle when the arm is abducted The affected arm swells and is cyanosed and dilated superficial veins appear on the arm in the axilla and over the anterior part of the chest The arm should be rested elevated and anticoagulant treatment given (see p 187)

### High Blood Pressure

**Definition** Increased blood pressure from any cause

**Physiology and Pathology** The maintenance of blood pressure depends mainly on two factors the cardiac output and the peripheral resistance Transitory changes in blood pressure may result from emotion pain exercise angina pectoris or a tabetic crisis The average blood pressure reading between the ages of 18 and 40 is 120/80 mm Hg Later in life the pressure may rise to 140/90 mm Hg A diastolic pressure permanently over 90 mm Hg in a young person is likely to be abnormal The pressure tends to rise in old age owing to loss of elasticity in the arteries Permanent high blood pressure is often discovered on routine examination without the patient having any symptoms

Rise of blood pressure may be due to 1 *Cardiac factors* An increased stroke output may be due to various conditions, such as an anxiety state aortic reflux or thyrotoxicosis Less often it results from adherent pericardium complete heart block patent ductus arteriosus or an arterio venous aneurysm 2 *Peripheral factors* The peripheral resistance varies with the condition of the arteries arterioles capillaries and veins and with the volume and viscosity of the blood Thickening and loss of elasticity of the arteries arteritis arteriolar thickening and spasm all tend to cause a rise of blood pressure Increased blood viscosity is met with in the polycythæmia hypertonica of Gaisbock

Hypertension may be acute or chronic *Acute hypertension* This is met with in acute glomerulonephritis and in the toxæmia of pregnancy *In both of these the rise of blood pressure is associated with* salt and water retention and in the majority of cases the hypertension is only temporary although occasionally it passes into a chronic phase

*Chronic hypertension* This may be due to 1 *Renal causes* These *are the most common known causes of hypertension including* chronic pyelonephritis chronic glomerulonephritis congenital polycystic disease of the kidneys congenital renal hypoplasia and some tumours Disturbance of the renal circulation causing ischæmia also results in high blood pressure This may be due to coarctation of the aorta to arteritis resulting from polyarteritis nodosa disseminated lupus erythematosus

or to thrombo angiitis obliterans It may also be associated with renal vein thrombosis renal artery aneurysm or hypoplasia of the renal artery

Chronic pyelonephritis is an important cause of hypertension in children and young people which may be easily overlooked as there may be nothing suggesting a renal lesion A retrograde pyelogram and catheterisation of the ureters with microscopical examination of the specimens obtained should be carried out in unexplained cases of hypertension in this age group

2 *Endocrine causes* *The suprarenal* Carcinoma adenoma or hyperplasia of the cortex result in salt and water retention From time to time in cases of phæochromocytoma (see p 698) there is a sudden outpouring of adrenaline and noradrenaline into the circulation causing a paroxysmal rise of blood pressure *The pituitary* In Cushing s disease the hypertension results from excessive production of cortico tropin (ACTH) stimulating the adrenal cortex The rise of blood pressure which may occur at the menopause is probably due to endocrine disturbances

## Essential Hypertension

(*Hyperpiesia*)

**Definition** A condition of hypertension of unknown origin usually not associated with demonstrable renal insufficiency

**Etiology** The cause is unknown Hypertension can be produced in dogs by partial constriction of one renal artery A pressor substance renin is thought to be produced in excessive amounts in the ischæmic kidneys Renin is thought to be formed or stored in the first con voluted tubules of the kidney and to combine in the blood with a substance called hypertensinogen (a globulin) to form a pressor substance called hypertensin This work suggests that essential hypertension is due to a toxic substance circulating in the blood and produced in the kidney whose blood supply is deficient Experi mentally removal of a kidney which has been rendered ischaemic is followed by a fall of blood pressure The high blood pressure does not result in any increase of general circulatory efficiency *Predisposing causes* 1 Age 30 to 50 years 2 Sex Females predominate slightly 3 Heredity There is a definite familial incidence

**Pathology** Post mortem examination usually reveals cardiac hypertrophy the left ventricle being affected Various arterioles in the body show arteriolosclerotic changes especially in the kidneys spleen pancreas liver and brain The kidneys may show granular changes with an adherent capsule

**Clinical Findings** Cases of essential hypertension fall into two groups according to the evolution of the disease they are mild or benign and severe or malignant *Benign hypertension* Here the onset is insidious the blood pressure being found to be raised at a routine examination without the patient being aware of any symptoms referable to it The blood pressure reading is often variable in the early stages later it becomes permanently raised After some years the

patient may complain of various symptoms, such as tinnitus headache palpitations, dyspnœa on exertion, giddiness lack of concentration irritability, numbness tingling cramp or coldness affecting the legs or transient visual disturbances *Severe hypertension* The severe symptoms often appear in the third or fourth decade A patient who has been known to have a high blood pressure for some years suddenly and comparatively rapidly deteriorates The main effect may fall on the heart the kidneys, the eyes or the brain

*On Examination* The physical signs depend upon whether the hypertension is benign or severe, as described below

**Course and Complications,** The course in benign hypertension is prolonged often for 10 or 20 years Death may eventually occur from cardiac failure cerebral hæmorrhage or from some intercurrent disease Women are less adversely affected by hypertension than are men In *severe hypertension* heart failure may rapidly ensue Hypertensive cerebral attacks are not uncommon In one form which is more prone to develop in patients under the age of 40 there is cerebral œdema, with severe headache vomiting drowsiness and possibly coma The retinal arterioles are constricted early in the attack The diastolic pressure may rise to 160 mm Hg The blood nitrogen figures are normal unless the kidney function has also failed The second variety is more common in individuals over the age of 40 and is probably due to angiospasm The patient suddenly becomes unconscious in an epilepti form attack There may be a preliminary cry This may be followed by coma or there may be transient aphasia, monoplegia or hemiplegia In other cases there are papillœdema retinal hæmorrhages and exudates and death ensues from cardiac or renal failure Cerebral hæmorrhage is a terminal event in other instances Probably about 55% of patients suffering from hypertension die from cardiac failure 35% from cerebral hæmorrhages or encephalopathy and 10% from renal failure

**Prognosis** This is difficult to assess in any individual case unless signs of malignant hypertension are present

**Treatment** In the early symptomless stages no strict regime is necessary Violent exercise should not be taken The patient should avoid over fatigue late hours and excess of tobacco alcohol or food No special diet is required When symptoms due to hypertension appear the treatment includes analgesics such as aspirin to relieve headache and the regular daily administration of tab phenobarb et theobrom (B P C) 1 mane a salt poor diet to reduce blood pressure various drugs to lower blood pressure and in some cases a surgical operation Kempner introduced the rice diet in 1944 for renal and hypertensive vascular disease The diet contains 250 to 350 G rice (dry weight) boiled in water or steamed The fluid is limited to 1 litre The diet is poor in protein and salt The fall in blood pressure is due to the low sodium content Increasing the sodium chloride even slightly causes the pressure to rise The diet is very unpalatable and is not without danger as uræmia may ensue This treatment is of little practical value It does however emphasise the value of restricting

the salt intake The sodium content of various articles of food is given on p 662 In many cases if a patient will keep steadfastly to a low salt diet the symptoms of hypertension provided they are not of the malignant variety can be relieved the blood pressure lowered and the health much improved

*Hypotensive drugs* The methonium salts produce their effect by blocking autonomic nerve ganglia The blockage of the sympathetic ganglia lowers the blood pressure but the parasympathetic ganglia are also affected which may result in dryness of the mouth blurred vision constipation impotence retention of urine etc

*Hexamethonium bromide* (Vegolysen) may be given by injection or orally best combined with reserpine It is preferable only to give it by injection The patient should be in bed and under medical observation in the early stages of treatment The initial dose for intramuscular or subcutaneous injection is mg 12 5 to mg 25 On the next day this dose can be repeated every 8 hours on the third day every 6 hours and on the fourth day every 4 hours the dose being gradually increased until about mg 500 are given in divided doses daily The drug is put up in 1% 2 5% 10% and 30% solutions for injection

*Pentapyrrolidinium bitartrate* (Ansolysen) is perhaps preferable to hexamethonium It is put up in mg 40 tablets The initial dose is mg 10 increased daily by mg 10 to 20 The amount required is usually between mg 20 to 100 a day but in some cases as much as mg 700 a day may be given It should be dissolved in water and taken half an hour before meals It may give rise to postural hypotension paralytic ileus difficulty in micturition dryness of the mouth or weakness of ocular accommodation

*Hydrallazine* (Apresoline) acts centrally It is given by mouth in doses of mg 25 every 4 to 6 hours It is not advisable to give this drug in cases of severe hypertension

*Rauwolfia serpentina* The purified alkaloid reserpine (Serpasil) is put up in mg 0 25 tablets This is given 3 to 4 times a day at the beginning of treatment and reduced to a maintenance dose of about mg 0 5 a day *Rauwiloid* is also an alkaloidal extract put up in mg 2 tablets It is given initially as 2 tablets daily taken at the same time reduced to 1 tablet daily The mode of action is uncertain

*Veriloid* This is an alkaloid derived from veratrum viride The dose is mg 2 four times a day

The most useful preparations for routine use in my experience are Ansolysen and Serpasil

*Surgical treatment* Bilateral splanchnicectomy may be beneficial in a small number of cases In the Smithwick operation the ganglionectomy is from D 9 to L 3 It is performed transdiaphragmatically in two stages The patient should be under the age of 50 The main indications are severe hypertension a blood pressure of 200/120 mm Hg or more which does not improve with rest and an absence of renal or myocardial disease or of generalised arteriosclerosis Retinal changes such as exudates and hæmorrhages are not contra indications in themselves and in successful cases they have proved to be reversible

Further the blood pressure should not be fixed, *i e* it should approximate to normal during sleep under light Pentothal Sodium anæsthesia, or with continuous caudal anæsthesia, the latter appears to be a more reliable test Thus the resting pressure before operation may be 200/130 with Pentothal Sodium 190/125, with caudal anæsthesia 140/90, and after sympathectomy 136/90 After the operation there may be a temporary postural hypotension and postural tachycardia The blood pressure is lowered permanently in about 15% of cases but the patient is often symptomatically better although the blood pressure remains raised

## Low Blood Pressure

*(Hypopiesis Subtension)*

**Definition** Diminished blood pressure from any cause

**Etiology** Low blood pressure may be physiological and unassociated with any symptoms of ill health It may be postural due to a failure of the blood pressure regulating mechanism as is seen in soldiers on parade standing for long periods at attention, or when patients take hexamethonium bromide to lower blood pressure Secondary hypotension may be met with in wasting diseases chronic infections and hypothyroidism It may be due to Addison s or Simmonds disease to hyperinsulinism, shock, hæmorrhage, coronary occlusion, neurocirculatory asthenia or to a hypersensitive carotid sinus

**Pathology** The low blood tension is usually due to capillary dilatation and in some cases the blood is pooled in the splanchnic area

**Clinical Findings** A low blood pressure may be an acute and temporary state which shows itself by fainting and loss of consciousness In other cases it is persistent, the patient complaining of lassitude insomnia headache and giddiness In some cases there are no symptoms as 8% of over 2 000 fit air pilots in England were found to be hypotensive

*On Examination* The blood pressure in an adult is considered to be low if the systolic reading is below 110 mm Hg

**Treatment** The blood pressure can be raised very temporarily by a subcutaneous injection of liq adrenal hydrochlor m 8 to 16 Ephedrine sulphate tablets gr ½ may be given by mouth t d s or amphetamine sulphate mg 10 to 20 daily in the morning A well fitting abdominal binder may be helpful

For anæmia iron or liver extract should be given according to the type of anæmia present Any of the associated conditions mentioned above require their special treatment

## Thrombo angiitis Obliterans

*(Thrombotic phlebo arteritis Buerger s Disease Von Winiwarter s Disease Die Hebraische Krankheit)*

**Definition** A disease characterised by thrombosis of portions of arteries and veins in the extremities with an inflammatory reaction around the affected vessels

**Etiology** The cause is unknown It is thought to be due to absorption of bacterial toxins, perhaps from the prostate or the intestines *It is not due to syphilis* *Predisposing causes* 1 *Age* 25 to 50 2 Sex Chiefly males 3 Heavy cigarette smoking 4 Race Chiefly Jews

**Pathology** Thrombosis occurs segmentally in the larger arteries of the legs and in the superficial veins There is some organisation of the clot and an inflammatory reaction around the vessels with fibrous tissue formation which may also involve the nerves

**Clinical Findings** The patient is usually a male between the ages of 25 and 50 years The onset often occurs in cold-weather with numbness coldness or pain in the foot or leg induced by walking and relieved by rest Later, the pain becomes more severe and occurs apart from exertion (rest pain) This indicates that the arterial blood supply and collateral circulation are grossly impaired and insufficient for the nutritional needs of the skin After a year or so the other leg is affected and later it may spread to the fingers and hands Sleep may become impossible owing to pain which is intensified when the leg is elevated Painful spots may also be noticed which are due to superficial venous thrombosis

*On Examination* In a developed case the leg is red and shiny when dependent and pale on elevation or on lying down no pulsation can be felt in the dorsalis pedis or posterior tibial arteries The foot or leg feels cold and the skin temperature is lower than that of the unaffected leg With an oscillometer accurate readings can be obtained of the difference in the blood pressure in the two legs the cuff can be placed above the knee and ankle Thrombosed superficial veins may be seen and felt their site varying from time to time There may be small blisters containing bloodstained fluid on the toes ulcers often form under the big toe nail and œdema of the foot or actual gangrene of the toes may ensue *There is no muscular wasting and no sensory changes are present* The determination of the skin temperature and the response to heat or to a spinal anæsthetic are of great importance in deciding the amount of vasoconstriction which can be overcome by sympathectomy They are an index of the amount of muscular tissue as compared with fibrous tissue in the arteries Alternatively they may indicate the degree to which the circulation is increased owing to vasodilatation of the healthy vessels The blanket method is a convenient way of performing the test The patient lies naked except for a loin cloth in a room with a constant temperature of 78° F for an hour The skin temperature is then taken with a special mercury thermometer or a thermocouple connected with a delicate galvanometer at the following points Mouth axilla elbow wrist interdigital spaces tips of digits groin knee ankle interdigital spaces tips of digits (on both sides of the body) The patient is then wrapped except for his face in a warmed light rubber sheet and covered by three blankets After one hour the skin temperatures are again taken at the same points and the results are recorded on a graph Normally or if there is little vasospasm the second graph is almost a straight line at or slightly below the mouth temperature level

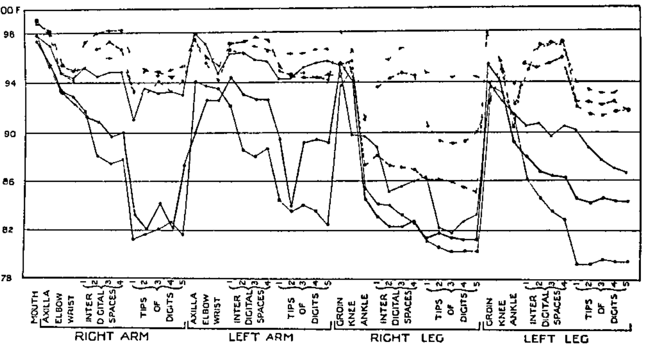
When a limb has arterial obstruction the skin temperature is some degrees lower than that of a normal limb and, on raising the temperature around the body the rise in skin temperature is proportional to the capacity of the superficial vessels to dilate (see Fig 17)

**Differential Diagnosis** The disease must be differentiated from Raynaud's disease erythromelalgia, arteriosclerotic endarteritis obliterans thrombo phlebitis migrans popliteal aneurysm, neuritis flat foot and rheumatism In Raynaud s disease there is no dependent rubor, no venous thrombosis and the arterial pulse is present In erythromelalgia there is no phlebitis no claudication no gangrene the arteries pulsate normally and the limb does not blanch markedly on elevation In arteriosclerotic endarteritis calcification of the arteries can be felt, or seen with the X rays there is no migrating phlebitis and the age incidence is usually later In thrombo phlebitis migrans there is no claudication and no arterial obliteration It is possible however that some cases are early stages of thrombo angiitis obliterans Many cases are diagnosed in the initial stages as flat foot or rheumatism and so much valuable time is lost

**Course and Complications** The course is usually prolonged for 10 years or longer, there is a tendency to spread from limb to limb but pulsation may return in the affected artery Thrombosis may occur in the mesenteric cerebral renal or coronary arteries Acute fulmination may occur with œdema and gangrene of the limb

**Prognosis** This is unfavourable death is usually due to an intercurrent disease

**Treatment** At the onset the patient should be kept in bed and *smoking forbidden Any septic focus should be treated* Local treatment consists in passive movements to the leg It is raised to produce blanching for 3 minutes and then hung over the edge of the bed to effect rubefaction for 5 minutes and finally it is kept horizontal for another 5 minutes This exercise is repeated several times daily For rest pains *the leg should be kept below the horizontal and relief is* obtained by hanging it down over the side of the bed Heat intensifies rest pains Vasodilator drugs may be tried such as nicotinic acid tab mg 50 in *daily doses sufficient to produce flushing and* tingling of the face neck back or hands As much as mg 500 a day may be required in divided *doses Priscol tab mg 25 or Cardophylin tab* 0 1 G may be taken by mouth 4 times daily Intravenous hypertonic saline injections are successful in some cases 300 ml of 3% NaCl solution being injected intravenously every other day As an alternative to this the patient may drink daily 3 to 4 quarts of water containing 10 G of table salt to 1 *quart Muscle extracts have proved beneficial in* the treatment of some cases a preparation such as Carnacton being used 1 to 2 ml are injected intramuscularly three times a day for several months The beneficial results of such treatment can be seen by the skin temperature curve in Fig 17 Anticoagulants (see p 187) may be used for an acute *thrombotic episode* Surgical treatment consists in lumbar sympathectomy with bilateral removal of the 2nd to 4th L ganglia The operation will only remove spasm and should therefore only be employed if the




Normal person after one hour at room temperature of 78 F
Normal person after wrapping in blanket for one hour at room temperature of 78 F
Patient after one hour at room temperature of 78 F
I atient after wrapping in blanket for one hour at room temperature of 78 F
I atient after two months Carnacton treatment and after one hour at room temperature of 78 F
I atient after two months Carnacton treatment and after wrapping in blanket for one hour at room temperature of 78 F

FIG 17 SKIN TEMPERATURES IN HEALTH AND IN THROMBO ANGIITIS OBLITERANS

[Tof ep .86

skin temperature tests indicate that the vessels are still capable of dilatation In cases of gangrene or pain unrelieved by other methods, amputation alone can afford relief

### Thrombo phlebitis Migrans

**Definition** Phlebitis of the superficial veins recurring in different sites and often accompanied or followed by venous thrombosis in internal organs

**Clinical Findings** Recurrent attacks of phlebitis occur usually in the legs The patient may have symptoms suggesting thrombosis in the lungs the abdomen the heart or the brain Thus he may be attacked with acute pain in the chest and cough up some rusty sputum or he may have severe abdominal pain or cardiac pain or symptoms of a cerebral thrombosis The temperature is usually elevated with each recurrence of thrombosis and the blood shows a leucocytosis

**Prognosis** This is usually favourable even when thrombosis occurs in internal organs although there is a tendency to recurrence

**Treatment** The patient must be kept in bed Plenty of fluids should be taken by mouth and a course of anticoagulants given (see p 187) Subsequently the teeth should be X rayed and any septic focus eradicated

### Erythromelalgia

*(Weir Mitchell s Disease)*

**Definition** A disease characterised by redness heat and pain of the extremities especially the feet which are worse when they are dependent

**Etiology** The cause is unknown It may be associated with exposure to cold and wet or with much walking

**Pathology** The vascular changes may be due to a disorder of the sympathetic nervous system

**Clinical Findings** The patient is usually an adult woman between the ages of 30 and 50 She first notices pain in the soles of the feet after standing or walking The pain is of a burning character and later may be very severe occurring also in bed (rest pain) when the feet are hot It is usually worse in the summer but there is no claudication

*On Examination* One or both feet are red and the lower half of the leg may also be affected The foot feels hot Pain is experienced when the surface temperature reaches 34 C There is no œdema and no gangrene The foot does not become much more red when dependent nor does it blanch considerably on elevation The arterial pulsation and sensation are normal

**Differential Diagnosis** Erythromelalgia must be differentiated from Raynaud s disease and painful rubor associated with thrombo angitis obliterans and arteriosclerosis affecting the extremities The differential diagnosis is considered on p 286

**Course and Complications** The disease is usually progressive but complications are rare Both hands and feet may eventually be affected

**Prognosis** The chance of spontaneous cure is remote and the disease usually persists for many years

**Treatment** Early and prolonged rest is of the greatest importance *Pain may be relieved by cold applications but if it is intense lumbar cord ganglionectomy is the most hopeful method of cure* The Wassermann reaction should be determined and if positive a course of anti syphilitic treatment is given

## Raynaud's Disease

**Definition** Raynaud s disease is a primary or idiopathic condition characterised by paroxysmal vasoconstriction of the arteries of the fingers, toes ears or nose which results in pallor or cyanosis with or without local gangrene These clinical changes are known as the Raynaud phenomenon

Secondary Raynaud's phenomenon is also met with In this similar clinical manifestations are present, but they are secondary to trauma to drugs or to organic disease affecting the arteries The causes are referred to below under the heading of differential diagnosis '

**Etiology** *Predisposing causes* 1 There is a familial incidence 2 Sex Females predominate 3 Age Usually between 20 and 40 years *Exciting causes* Exposure to cold and emotional disturbances

**Pathology** In the early stages no changes are found in the digital arteries later there may be thrombotic occlusion There are two theories as to the pathogenesis (*a*) the vasoconstriction is due to sympathetic nerve stimuli (*b*) the arteries are locally unduly susceptible to cold

**Clinical Findings** The patient is usually a female between the ages of 10 and 30 years although many cases have been recorded in males In a mild case (local syncope) the fingers and toes become suddenly pale and dead and on warming them they ache or throb The spasm spreads upwards from the tips of the fingers and toes In a moderately severe case (local asphyxia) the patient complains of a tingling sensation in the hands when they are cold and she may drop what she is holding If the white finger is immersed in warm water it goes blue One or more fingers go quite blue or black and there is severe pain The position of the hands does not influence the colour of the fingers in the attacks The ears or nose may be similarly affected In a severe case (local gangrene) the pain is almost unbearable and gangrene with sloughing may occur in the fingers, toes ears or nose The affected parts may

Raynaud s phenomenon may be associated with a variety of causes which include local trauma vibration from pneumatic drills ergot poisoning collagen diseases such as disseminated lupus erythematosus and dermatomyositis thrombo angiitis obliterans arteriosclerosis cold hæmagglutination polycythæmia etc

**Course and Complications** The course is usually progressive for a time Trophic changes which may develop in severe cases include sclerodactyly ridging of the nails blister formation and small local areas of gangrene in the finger tips More extensive gangrene is uncommon

**Prognosis** This is variable, but the condition may disappear after several years

**Treatment** *Prophylactic* In the secondary Raynaud s phenomenon due to pneumatic drills the workers should have their hands protected by sorbo shock absorbing pads placed in the palms of their gauntlets The use of gloves or rubber pads interferes with the accurate control of more delicate instruments In view of the long incubation period it has been suggested that no one should be employed in this type of work for longer than 9 months

*Curative* The extremities should be protected from cold A warm climate is very beneficial Smoking is forbidden Priscol tablets mg 25 may be taken 2 tablets 4 times daily Massage and electrical treat ment with galvanism or high frequency should be tried In severe cases affecting the lower limbs the operation of lumbar sympathectomy may afford a cure if the vessels are still capable of dilatation When the upper limbs are affected the results of excision of the stellate ganglion in which post ganglionic fibres are divided are not so good as in lumbar sympathectomy in which pre ganglionic fibres only are divided A pre ganglionic sympathectomy in which the sympathetic trunk is divided below the third thoracic ganglion and the second and third thoracic somatic nerves are divided at the vertebral foramina is pre ferable therefore to stellate ganglionectomy and avoids the development of a Horner s syndrome

## Angio neurotic Œdema

### (*Quincke s Disease*)

**Definition** A condition characterised by paroxysmal attacks of œdematous swelling of the skin subcutaneous tissues or mucous membranes

**Etiology** The cause is not known It may be an allergic mani festation or a vaso motor disturbance It may occur as an allergic reaction to penicillin

**Pathology** The œdema may result from the local effect of histamine on capillaries

**Clinical Findings** The patient is usually a young adult of either sex and a history is sometimes obtained of similar attacks in other members of the family The most serious form with œdema of the glottis is often hereditary He complains of localised swellings appear

ing suddenly in the face lips, eyelids mouth, hands or elsewhere urticarial rash may develop simultaneously The eyelids or lips swell and become very tense and painful Thus the eyes ma completely closed Similar swellings may develop in the tongu larynx causing urgent dyspnœa The mucous membrane of alimentary tract may be affected, the patient complaining of a abdominal pain with vomiting or diarrhœa

*On Examination* The appearance of the patient may be compl altered during the attacks owing to the swelling The cutaneous s ings pit slightly on pressure the skin is generally white but may little pink

**Differential Diagnosis** Other varieties of œdema, such as produced by disease of the heart or kidneys must be excluded abdominal variety is suggestive of an acute abdominal lesion, but t is no fever and a history of previous attacks affecting the skin i afford the clue to the diagnosis

**Course and Complications** Recurrent attacks are to be expec Hæmoglobinuria may be a complication

**Prognosis** The patient often outgrows the attacks Death i ensue from œdema of the glottis

**Treatment** *During the attack* An injection of m 8 to 16 of adrenal hydrochlor should be given subcutaneously Antihistan drugs such as Thephorin tab mg 25 or Pyribenzamine mg 50 may taken by mouth two or three daily In severe cases cortisone may *given by mouth beginning with mg 200 a day in divided doses* reducing gradually to mg 25 or 50 daily With œdema of the glott nebuliser containing 1/100 adrenaline hydrochloride should be us Tracheotomy or intubation of the larynx may be necessary to s life *Between the attacks* A search should be made for septic f in such sites as the teeth tonsils, antra, sinuses intestines kidne bladder and genital organs Any such focus should be adequat treated The protein cutaneous tests should also be performed T effect of different diets should be determined eliminating such artic as milk eggs or fish

### Milroy's Disease

*(Hereditary Trophœdema Non filarial Elephantiasis)*

**Definition** A disease which is often familial characterised by swe ing of the legs arms or face

**Etiology** *The cause is not known It is more common in wom* than in men and may be noted shortly after birth or not be appare until after the age of puberty

**Pathology** There is thickening of the skin and subcutaneo tissues

**Clinical Findings** The patient may say that other members of h family are similarly affected She complains of heaviness and swelli of the feet and legs Only one limb may be affected It is uncomm for the arms or face to be involved

*On Examination* The affected limb is usually pale swollen and pits slightly on pressure

**Differential Diagnosis** Other causes of swelling of the extremities must be excluded such as diseases of the heart or kidneys varicose veins obesity intra abdominal venous obstruction or lymphatic obstruction due to filarial elephantiasis (see p 759)

**Course and Complications** The course is progressive but it may be interrupted by acute attacks of pain in the limbs accompanied by fever

**Prognosis** The disease is neither fatal nor can it often be cured

**Treatment** The legs should be bandaged from the feet upwards to prevent the swelling from increasing Kondoléon's operation is sometimes performed with satisfactory results Strips of œdematous skin subcutaneous tissue and deep fascia are removed from both sides of the leg from the upper end of the thigh to the ankle with an interval of about 3 inches on each side of the knee

## CHAPTER IV

## THE NERVOUS SYSTEM

**Introductory** Every patient who is suspected to be suffering from a disorder of the nervous system should be examined in accordance with a definite plan as detailed later (see p 298) An elementary knowledge of the anatomy and physiology of the nervous system is essential

### Anatomy and Physiology

**The Motor Path from the Cerebral Cortex to the Muscles** Voluntary muscular movements are initiated by impulses arising in the cerebral cortex, and passing to the mid brain pons medulla or spinal cord From these lower levels fresh fibres arise and pass direct to the muscles concerned It is believed that there are three relays of fibres between the cerebral cortex and the muscles as described below The motor cortical area lies in the frontal lobe of the brain on each side just anterior to the fissure of Rolando (see Fig 30) The motor fibres converge and pass through the genu and anterior two thirds of the posterior limb of the internal capsule of the brain This is the portion of white matter which lies between the caudate nucleus and optic thalamus on its inner side, and the lenticular nucleus on its outer side It will be noticed (see Fig 18) that in the convergent movement the fibres for the leg cross those of the arm and come to lie posteriorly whereas the face fibres also cross the arm ones to become anterior the arm fibres being situated centrally The order of fibres in the internal capsule is therefore from before backwards face arm, leg

The upper motor neurones emerge from the internal capsule and run through the mid brain, pons and medulla, near the ventral surface in a bundle called the pyramid Certain cranial motor nerves have their cells of origin in this part of the brain Thus the nucleus of the III nerve lies at the level of the superior corpora quadrigemina (see Fig 34) and that of the IV nerve in the region of the inferior corpora quadrigemina (see Fig 35) The motor nucleus of the V nerve is in the middle of the pons and the VI and VII nuclei in the lower part of the pons The IX X XI and XII nuclei are in the medulla (see Fig 42) It will be noticed that with the exception of the III and IV nerves the cranial nerves pass to the same side of the body as that on which their nuclei are situated Decussation of certain fibres occurs with the III nerve and total decussation in the case of the IV nerve so that the nucleus of the IV nerve situated on the right side of the brain supplies the left superior oblique muscle, and *vice versâ* There is a bilateral cortical innervation for all the cranial nerves except the XII and the part of the VII nerve which supplies the lower part of the face As the upper neurones which constitute the pyramidal tract, pass down through the mid brain pons and medulla they give off

fibres which cross the mid line and end in arborisations in grey matter from which the second relay connects with the dendrites of the motor cranial nerve nuclei

On tracing the pyramidal tract still lower there is at the level of the lower part of the medulla a decussation affecting 90% of the fibres The fibres after decussation are known as the crossed pyramidal tract and they run down the spinal cord in the lateral regions (see

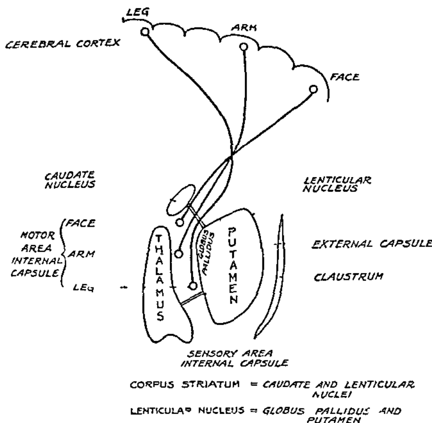



FIG 18 DIAGRAM SHOWING THE INTERNAL CAPSULE AND CORPUS STRIATUM

Fig 19) The 10% of fibres which have not decussated form the direct pyramidal tract and pass down the anterior part of the cord near the median fissure as far as the lower cervical or mid thoracic region (see Fig 19) The fibres of the crossed pyramidal tract are connected with posterior horn cells and then by a second relay with the motor cells in the anterior horn of the grey matter of the spinal cord These are the cells of origin of the lower motor neurones from which nerve fibres proceed direct to the skeletal muscles The upper motor neurones in the direct pyramidal tracts cross the mid line in the anterior white commissure of the cord and terminate around posterior horn cells

They are connected by a second relay with anterior horn cells There are thus three relays between the cerebral cortex and the muscles, the upper motor neurones, the intermediate neurones, and the lower motor neurones

**The Extra pyramidal Motor Tracts** (The striato spinal motor system ) These tracts convey involuntary motor impulses from various ganglia in the brain to intermediate relay stations or to the anterior horn cells of the cord They are concerned with automatic actions and are an older motor system than is the pyramidal one They function in infants before the pyramidal system is capable of so doing The most important of these tracts are —

1 *The Vestibulo spinal Tract* The fibres arise in the lateral

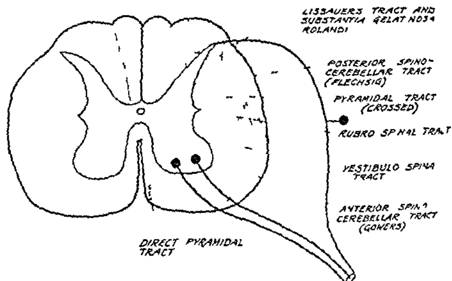



Fig 19 Diagram of Section of Spinal Cord

vestibular nucleus of the VIII nerve (Deiters' nucleus) in the lower part of the pons and run down the antero lateral part of the medulla and cord to end around anterior horn cells on the same side of the cord (see Fig 31)

2 *The Tecto spinal Tract* This tract arises in the mid brain at the level of the superior corpora quadrigemina, and, after crossing in Meynert s or the fountain decussation it passes down the cord in the anterior longitudinal bundle to end around anterior horn cells It conveys impulses to the voluntary muscles (eye protection movements) as the result of stimuli from the eyes which have passed to the calcarine fissure and thence to the superior corpora quadrigemina (see Fig 34)

3 *The Cerebello rubral Tract* The fibres arise in the lateral lobe of the cerebellum, and pass by the superior cerebellar peduncle to the red nucleus of the opposite side (see Fig 31)

4 *The Striato rubral Tract* (The large celled pallidal system ) The fibres arise in the globus pallidus of the lenticular nucleus and run to the red nucleus on the same side of the brain The impulses are concerned with emotional and associated movements

5 *The Small celled Neo striate System* Fibres pass from the caudate nucleus and the putamen of the lenticular nucleus to the globus pallidus They exert an inhibitory influence over the motor impulses arising in the large celled pallidal system

6 *The Rubro spinal Tract* The fibres arise in the red nucleus which is situated in the mid brain at the level of the superior corpora quadrigemina The fibres immediately decussate and pass down the opposite side through the pons and medulla and antero lateral part of the cord to end around anterior horn cells They convey impulses for group movements (see Fig 31)

**Upper Motor Neurone Lesions** Irritative lesions cause convulsive movements of the voluntary muscles

Paralytic lesions result in paralysis of voluntary movement with spasticity increase of deep reflexes ankle and patellar clonus and an extensor plantar response The electrical reactions are normal and wasting is slight When the upper motor neurones arising in the cerebral cortex are put out of action the extra pyramidal motor tracts arising in the basal ganglia may show symptoms of uncontrolled activity which are known as release symptoms Various involuntary movements then ensue such as are seen clinically in athetosis following a hemiplegia

**Extra pyramidal Motor Neurone Lesions** Lesions of the large celled pallidal system (globus pallidus) as occur in paralysis agitans and progressive lenticular degeneration cause hypertonus of muscles tremors and disturbance of automatic movements Lesions of the small celled neo striate system produce athetoid or choreic movements Lower motor neurone lesions result in weakness of muscles wasting hypotonus absence of the deep reflexes and a reaction of degeneration

**The Sensory Path from the Periphery to the Brain** 1 *The Spinal Nerves* In the spinal motor nerves about 40% of the fibres are afferent conveying sensations which are known as proprioceptive or kinæsthetic from the muscles tendons joints and periosteum Their cells of origin are situated in the posterior root ganglia The spinal sensory nerves convey impulses of pain touch and temperature from the skin These are called exteroceptive impulses Enteroceptive impulses pass from the viscera by various spinal nerve roots and connect with cells in the lateral horn of the grey matter of the cord The following impulses enter the spinal cord by various posterior root fibres (see Fig 20) 1 Touch (deep cutaneous) and discrimination of the points of a compass 2 Muscle sense as evidenced by judging the differences in weight stereognosis or judging the nature of objects by feeling them deep pressure pain tendon sense joint sense bone vibration sense and the knowledge of the position of the limb in space 3 and 4 Muscle tonus equilibrium and co ordination 5 Cutaneous pain and temperature 6 Localised touch 7 Direct cutaneous—muscle reflex path

1, 2, 3 and 4 are kinæsthetic sensations 5 and 6 are skin sensations

2 *The Central Path* (see Figs 20, 21) *Impulses* 1 *and* 2 The fibres conveying deep touch discrimination of compass points muscle tendon joint and bone sense pass up the cord on the same side in the posterior column of Burdach As they ascend they are pushed centrally

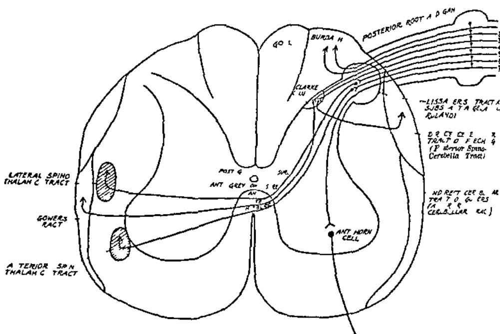


FIG 20 DIAGRAM SHOWING FIBRES OF A POSTERIOR NERVE ROOT ENTERING THE CORD

*Posterior Root Tracts*—

1 Touch (deep cutaneous) Discrimination of points of compass
2 Muscle sense Joint sense Tendon sense Bone vibration sense Pressure-pain of muscles
3 and 4 Muscle tonus Equilibrium Co ordination
} Kinæsthetic stimuli
5 Pain Temperature
6 Localised touch
7 Direct cutaneous muscle reflex

*Posterior columns* *Muscle sense* such as sense of position of a limb judging weights pressure pain
*Stereognostic sense* a combination of touch and joint sense
*Joint sense* (position of a joint)
*Bone vibration sense*
*Tactile* discrimination of two points Sense of position of skin when pulled out.
*Lateral Spino thalamic Tract* Pain and temperature
*Anterior Spino thalamic Tract* Highly localised touch
*Spino cerebellar Tracts* (Gowers and direct cerebellar) Muscle tonus

by new fibres coming in and so they constitute the column of Goll The fibres terminate in the gracile and cuneate nuclei in the lower part of the medulla The gracile nucleus is the relay station for fibres from the leg and the cuneate nucleus for the fibres from the arm The Th 3–12 nerves do not appear to have any connection with the gracile or cuneate nuclei A second relay of fibres now begins which decussates (decussation of the fillet) and then ascends the pons and mid brain as

the mesial fillet to the antero lateral nucleus of the optic tha
From the optic thalamus some impulses pass by a third relay
sensory cerebral cortex in the posterior central gyrus behin
Rolandic fissure Other fibres pass from the gracile and cuneate
to the cerebellum by the superficial and deep arcuate fibres whi
into the inferior cerebellar peduncle (see Fig 81)

*Impulses 3 and 4* Muscle tonus sensations and impulses for
taining equilibrium relay first in Clarke s column near the po

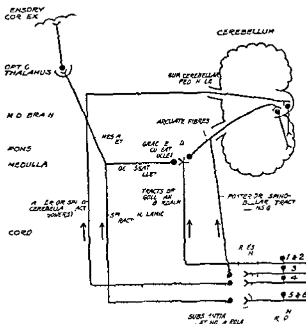



FIG 21 DIAGRAM SHOWING THE SENSORY PATH

*Fibres in Posterior Root—*
1 & 2 Kinæsthetic
3 & 4 Equilibrium
5 & 6 Pain Temperature and some touch sensations

horn of grey matter The second relay of fibres conveys the im
either to the direct cerebellar tract of Flechsig (posterior spino cer
tract) on the same side or cross the mid line in the grey or the a
white commissure to the indirect cerebellar tract of Gowers (a
spino cerebellar tract) on the other side The direct cerebellar
conveys the impulses to the cerebellum on the same side by the i
cerebellar peduncle whereas the indirect cerebellar tract dec
higher up (see p 370) and passes to the cerebellum by the su
cerebellar peduncle on the same side as that on which the im
entered the cord

*Impulses 5 and 6* Sensations of pain and temperature enter th

by the posterior root and relay in the substantia gelatinosa Rolandi The second relay conveys the impulses to the opposite side of the cord through the anterior white commissure and they then ascend in the lateral part of the spino thalamic tract This joins the mesial fillet and runs to the antero lateral nucleus of the thalamus Some of the impulses are then carried by a third relay to the sensory part of the cerebral cortex (the post central gyrus, the superior parietal lobule the supramarginal and angular gyri) and others pass to the mesial thalamic nucleus (the essential thalamic organ) where they give rise to sensations of pleasure and pain The fibres conveying the highly localised sense of touch also relay in the substantia gelatinosa Rolandi The second relay crosses the mid line in the anterior white commissure and ascends in the anterior part of the spino thalamic tract These fibres join the mesial fillet, and terminate in the optic thalamus A final relay conveys the impulses to the sensory part of the cerebral cortex

**The Cranial Nerves** (see p 375) The sensory fibres from the V, VII IX and X nerves also join the fillet

**The Cerebellar Connections** (see p 368)

**Nervous Disease Case Sheet**

Name Age Sex M S W
Family history of nervous diseases
Previous history
History of present illness Date of onset
Mode of onset
General condition Pulse Temperature
Respirations Urine Blood (cell count and W R ) Cerebration Speech

**Cranial Nerves** I
II
III IV VI
V Motor Sensory
VII Motor Sensory
VIII
IX X and accessory part of XI Motor Sensory
XI Spinal part
XII

**Spinal Motor Nerves** (*a*) Power of muscles
(*b*) Wasting or hypertrophy of muscles
(*c*) Tone of muscles
(*d*) Tremors, fibrillations, spasms or convulsions

**Spinal Sensory Nerves** (*a*) Tactile discrimination (compass test)
(*b*) Muscle sense
(*c*) Stereognostic sense
(*d*) Muscle and tendon pressure sense

(e) Joint sense
(f) Vibration sense
(g) Pain temperature and light touch
(h) Subjective sensation

**The Reflexes** (a) *Superficial*

1 Conjunctival (V)
2 Palatal (IX and X)
3 Pharyngeal (IX)
4 Epigastric (Th 7–9)
5 Abdominal (Th 9–12)
6 Cremasteric (L 1 and 2)
7 Plantar (S 1 and 2)

(b) *Deep*

1 Pupil (II and III)
2 Jaw jerk (V)
3 Biceps (C 5 and 6)
4 Triceps (C 6 and 7)
5 Supinator (C 7 and 8)
6 Knee jerk and clonus (L 2–4)
7 Ankle jerk and clonus (S 1 and 2)

(c) *Visceral*

1 The bladder
2 The rectum

**Co ordination** (a) The upper limbs
(b) The lower limbs

**Trophic Changes.** (a) Skin
(b) Bones and joints

**Gait**
**Electrical Reactions**
**Electromyography**
**Lumbar Puncture**
**Myelography**
**Ventriculography**
**Encephalography**
**Angiography**
**Electro Encephalography**
**Site of Lesion**

**Additional notes on the examination of the nervous system** —The chief segmental nerve supply for some of the important muscles is as follows C 4 Diaphragm Trapezius C 5 *Supraspinatus Infra spinatus* Biceps Deltoid C 6 Pronators of forearm Latissimus dorsi

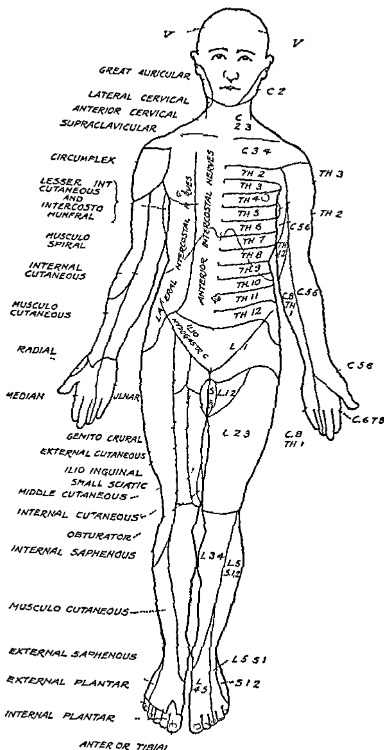



FIG 22A THE AREAS OF SKIN SUPPLIED BY THE SPINAL NERVES ARE SHOWN IN THE LEFT HALF OF THE FIGURE AND THE SKIN AREAS SUPPLIED BY THE PERIPHERAL NERVES ARE INDICATED IN THE RIGHT HALF OF THE FIGURE (After Cunningham)

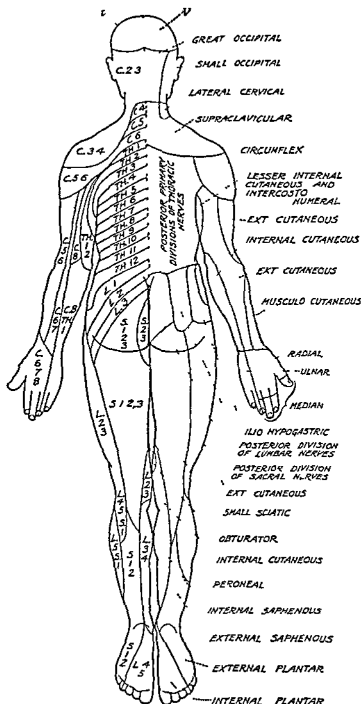



FIG 2°B THE AREAS OF SKIN SUPPLIED BY THE SPINAL NERVES ARE SHOWN IN THE LEFT HALF OF THE FIGURE AND THE SKIN AREAS SUPPLIED BY THE PERIPHERAL NERVES ARE INDICATED IN THE RIGHT HALF OF THE FIGURE (After Cunningham)

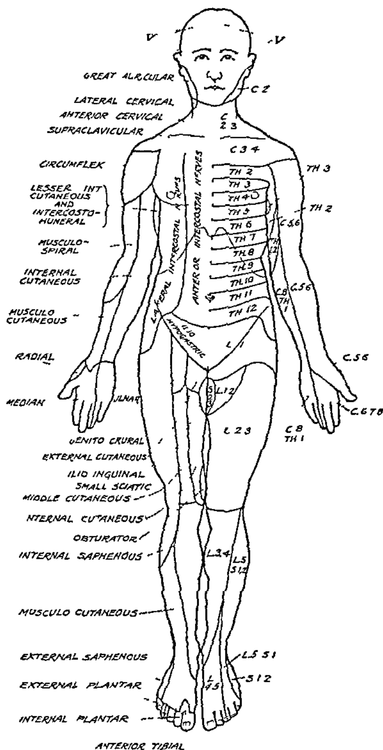



FIG 2 A. THE AREAS OF SKIN SUPPLIED BY THE SPINAL NERVES ARE SHOWN IN THE LEFT HALF OF THE FIGURE AND THE SKIN AREAS SUPPLIED BY THE PERIPHERAL NERVES ARE INDICATED IN THE RIGHT HALF OF THE FIGURE (After Cunningham.)

of the skull the presence of bossing the bony texture the presence of fracture the state of the sutures the presence of erosion of the dorsum sellæ or clinoid processes the vascular markings the presence of abnormal or normal calcifications and the size of the optic foramina, etc

**Ventriculography** (see p 334)

**Encephalography** (see p 334)

**Angiography** For lateral views 10 ml of 35% diodone are injected into the internal carotid artery and X ray films of the skull are taken during the injection 2 seconds later and again 2 seconds later For antero posterior views 10 ml of a 42% solution of diodone are used This method assists in the diagnosis of aneurysms cerebral hæmorrhage and cerebral tumours

**Electro encephalography** Changes in electric potential arising in the cerebral cortex can be demonstrated with electrodes applied to the scalp The normal electro encephalogram shows characteristically







FIG 23 NORMAL ELECTRO ENCEPHALOGRAM AND TYPES OF WAVES IN EPILEPSY LENNOX *Jour Amer Med Assocn*

small alpha waves With a cerebral tumour or abscess and in epilepsy abnormal waves may be seen (see Fig 23 and pp 327 346)

## MENINGITIS

**Definition** Inflammation of the meninges

*Varieties* 1 *Pachymeningitis* is inflammation of the dura mater This is often secondary to disease or injury of the bone such as spinal caries or a fractured skull Hæmorrhagic pachymeningitis is met with in general paralysis of the insane In chronic alcoholism and senile dementia it probably results from slight injuries to the skull not noticed by the patient Pachymeningitis cervicalis hypertrophica may be due to syphilis and less frequently to tuberculosis

2 *Leptomeningitis implies inflammation of the* pia arachnoid There is often also ependymitis or inflammation of the lining membrane of the cerebral ventricles Leptomeningitis is usually due to invasion of the cerebro spinal fluid by micro organisms such as the Mycobacterium tuberculosis (B tuberculosis) the Neisseria meningitidis (meningococcus) the Diplococcus pneumoniæ (pneumococcus) the streptococcus the staphylococcus the Neisseria gonorrhœæ (gonococcus) the Bacterium typhosum the Bact coli the Treponema pallidum and less often the Hæmophilus influenzæ Leptospiral meningitis is due either to the Leptospira icterohæmorrhagiæ or to the L canicola Aseptic meningitis and serous meningitis are also described

C 7 Triceps Extensors of wrist and fingers C 8 Flexors of wrist and fingers Th 1 Intrinsic muscles of the hand Th 2–10 Intercostals Th 7–12 Abdominal wall muscles L 1 Quadratus lumborum L 3 Adductors of thigh Iliopsoas L 4 Extensors of knee and abductors of thigh L 5 Flexors of knee S 1 Glutei and calf muscles S 2 Anterior tibial muscles Peronei Intrinsic muscles of foot S 3 and 4 Pelvic muscles The cutaneous peripheral nerve and nerve root areas are illustrated in Fig 22

**Co ordination** (*a*) Upper limbs This is tested by the ability to pick up small objects and to touch the tip of the nose with the eyes shut

(*b*) Lower limbs Romberg s sign The patient cannot stand with the feet together and eyes closed Co ordination is tested also by asking the patient while lying down to touch with his toe an object held near it, or to touch his knee with the opposite heel

(*c*) Past pointing This test is described on p 398

**Trophic Changes** *Skin e g* glossy skin perforating ulcers painless whitlows herpes zoster bed sores leucoderma, and increased sweating *Bones and joints, e g* facial hemiatrophy or hemihypertrophy inequality of the two halves of the body arthropathies

**The Gait** Spastic ataxic cerebellar, scissors, high stepping reeling festinating hysterical etc

**The Electrical Reactions** The stimulus is applied at the motor point where the nerve enters the muscle Normally muscle directly stimulated responds to the make and break of a galvanic current but not to a faradic stimulation If the muscle is stimulated through the nerve at the motor point it normally responds to faradisation and also to the make and break of the galvanic current

*Reaction of Degeneration* The muscle when stimulated at the motor point, does not respond to faradic stimulation owing to nerve degeneration and the response to galvanism is modified so that it is sluggish or the anodal closure contraction is greater than the kathodal closure contraction *i e* A C C 7 K C C Normally the reverse is the case

**Electromyography** This affords more accurate information concerning the state of the peripheral nerves and the voluntary muscles than is given by the reaction of degeneration Needle electrodes are inserted in the muscle and changes in electric potentials during contraction are amplified by valves and recorded by a cathode ray oscillograph

**Lumbar Puncture** Normal cerebro spinal fluid Pressure 150 mm $H_2O$ Cells 1 to 5 mononuclears per cmm Protein 20 to 40 mg urea and non protein nitrogen 15 to 30 mg chlorides 700 to 750 mg sugar 70 to 100 mg per 100 ml Lange curve 00000000000

**Myelography** By injecting a radio opaque substance such as Pantopaque into the cisterna magna or into the theca by lumbar puncture the site of the spinal cord obstruction can be visualised by X ray examinations

In the diagnosis and localisation of intracranial tumours certain other investigations may be required These include —

**Radiography** The skull must be X rayed from several angles By this means information is obtained as regards the size and shape

hips and knees (Brudzinski s sign) The temperature is raised to 101° or 103° F and the pulse is frequent such as 100 to 120 The abdomen is rather scaphoid, the bowels are constipated and vomiting occurs apart from taking food (cerebral vomiting) The deep reflexes are usually exaggerated This is the *stage of irritation*

A few days later a *stage of compression* may be reached The patient is now definitely drowsy or comatose but localised or general muscular convulsions may be seen with irregular facial contractions There is more marked head retraction but the back is rarely arched The pupils are dilated and often unequal and the patient may complain of difficulty in reading In infants the anterior fontanelle may bulge Optic neuritis and less often choroidal tubercles may be found on ophthalmoscopic examination The pulse is slower and premature systoles may be noted the temperature is often lower about 99 in the evening

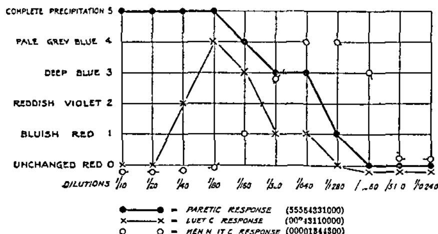



FIG 24 LANGE COLLOIDAL GOLD CURVES

Vomiting usually ceases during this stage Later still a *paralytic stage* may be reached with incontinence of fæces and urine and a low temperature Vomiting may now recur The pulse tends to be more frequent and is often very rapid shortly before the end Just before death the temperature may rise to 106° F or higher The pupils are dilated and do not react to light The anterior fontanelle now becomes flat The child lies on his back with the legs extended The abdominal and deep reflexes are abolished In many cases it is not possible to differentiate these stages The Mantoux reaction is positive in about 85% of cases The cerebro spinal fluid The fluid is under pressure clear or slightly turbid On standing a fine coagulum often forms Tubercle bacilli may be found in the majority of cases in the coagulum Lymphocytes are present in excess up to 400 or more per c mm In the early stages however 50 or 60% of the cells may be polymorphonuclears There is an excess of protein and the Nonne Apelt test for globulin is positive Sugar is diminished to 15 or 20 mg per 100 ml and

8 *Arachnoiditis* is inflammation of the layers of the arachnoid with cyst formation

### Tuberculous Meningitis

### (*Basilar Meningitis*)

**Etiology** The disease is caused by the Mycobacterium tuberculosis (B tuberculosis) 75% being of the human and 25% of the bovine type *Predisposing causes* 1 Age Usually children between the ages of 1 and 6 Adults may be affected 2 Sex Equal *incidence*

**Pathology** Tuberculous meningitis is usually secondary to a tuberculous focus in the brain The organisms are frequently carried to the brain by the blood stream from a tuberculous gland in the chest or abdomen In some cases the brain lesions are secondary to tuberculous foci in the lungs bones joints skin or genito urinary organs The development of meningitis is thus a fortuitous event and does not depend on the existence of miliary tuberculosis An operation on a tuberculous joint may cause a generalisation of the tuberculous infection with resultant meningitis In adults tuberculous meningitis is usually a terminal event in cases of pulmonary or genito urinary tuberculosis At autopsy there is inflammation of the pia at the base of the brain especially in the interpeduncular space the Sylvian fissure and optic chiasma Minute tubercles may be seen on the pia or on the branches of the middle cerebral anterior and posterior perforating arteries The cerebral convolutions are somewhat flattened the brain substance softened, and the ventricles contain an excess of cerebro spinal fluid producing a moderate degree of hydrocephalus Minute tubercles may be found in other parts of the body such as the lungs, liver and spleen if a condition of miliary tuberculosis exists

**Clinical Findings** The patient is often a child of about 8 years of age In some cases there is a history of a blow on the head some weeks previously or of a recent attack of measles or of whooping cough In other instances the child has been infected by a person in the home suffering from pulmonary tuberculosis with tubercle bacilli in the sputum Often the patient has been apparently in good health but for a few weeks previously has become listless and irritable with headache loss of appetite slight temperature and loss of weight The first symptom noted may be a convulsion

*On Examination* In the early stages the patient is usually rather irritable restless and does not like being examined drawing up the bed clothes over himself (Stocker s sign) He may cry out from time to time ( hydrocephalic cry ) and is easily startled by noises He lies on his side with the knees drawn up Twitching of various muscles may be seen and there may be photophobia The only signs of an organic lesion which may be found are strabismus due usually to weakness of the external rectus muscle, ptosis and some rigidity of the neck and back muscles The pupils are rather small and the child may grind his teeth Flexion of the hip with the knee extended is resisted (Kernig s sign) and similarly extension of the shoulder with the elbow extended may cause pain (Bikele s sign) *Flexion of the neck causes flexion of one or both*

## Meningococcal Meningitis

*(including Posterior Basic Meningitis of Infants Cerebro spinal Fever Spotted Fever)*

**Etiology** The disease is due to the Neisseria meningitidis (meningococcus) of which there are four serological types Group I (types I and III) and Group II (types II and IV) *Predisposing causes* 1 Age Infants young children and young adults 2 Sex Incidence equal but males predominate in war time 3 Season Winter and spring 4 Lack of ventilation and overcrowding at night 5 Fatigue 6 Naso pharyngeal catarrh 7 Climate World wide distribution Sporadic cases and epidemics occur Infection is spread by ' droplets from carriers rarely from patients Carriers are usually temporary ones for periods up to 3 weeks chronic carriers are rare

**Pathology** The meningococcus probably spreads from the naso pharynx by the blood stream to the brain and spinal cord Petechial hæmorrhages occur in the brain and spinal cord and it is suggested that organisms present in these hæmorrhages pass into the cerebro spinal fluid The pia arachnoid at the base of the brain is inflamed around the pons medulla and under surface of the cerebellum An exudate occurs between these membranes The meninges covering the vertex of the brain and the cord are inflamed and encephalitis is present The lateral ventricles may be dilated giving rise to hydro cephalus Purulent exudate may be present around the cord chiefly on the posterior aspect in the lumbar region The mesenteric glands and intestinal lymphoid tissue are often swollen and the sphenoidal sinus may contain pus Suprarenal hæmorrhage occurs in some fulminating cases and encephalitis in others

**Incubation Period** 1 to 8 days

**Clinical Findings** The disease usually begins suddenly with a rigor severe headache (occipital) vomiting and in young children convulsions

*Early or Septicæmic Stage* This usually lasts about 48 hours but it may be considerably shorter The temperature rises rapidly to 102 or 103° F There is pain in the head back and legs Petechial or purpuric rashes may be seen usually on the trunk or inner sides of the thighs about the second or third day of the disease A blotchy erythema may also be noted chiefly on the extremities Painful symmetrical arthritis may affect various joints usually without effusion

*Meningeal Stage* This usually rapidly ensues There is stiffness in the neck passing on to rigidity with some degree of head retraction the patient lying on his side with hips and knees flexed He is apathetic the face somewhat flushed the pupils are usually equal and dilated reacting sluggishly to light The vomiting persists and the headache becomes very severe occurring in paroxysms which cause him to scream with pain The patient complains of hyperæsthesia of the skin to touch and cold and also of photophobia

The most important physical signs are the absence of the abdominal reflex the presence of Kernig s sign (inability to extend fully the knee when the hip joint is flexed at a right angle) neck rigidity and Brud

the chlorides are reduced to 500 or 550 mg per 100 ml The Lange test shows a meningitic curve (see Fig 24) The blood A leucocytosis of about 12 000 per c mm may be present

**Differential Diagnosis** In the early stages the illness may be mistaken for influenza or gastritis Thus headache vomiting drowsiness fever, and convulsions may all occur in gastro enteritis Apical pneumonia in a child may at the onset closely simulate meningitis In meningism there are symptoms and signs of meningitis although the results of lumbar puncture show no abnormality The prolonged fever may suggest typhoid fever The nervous symptoms may be confused with polioencephalitis or with encephalitis lethargica A cerebral tumour or abscess may be mistaken for tuberculous meningitis, but in the former optic neuritis is usually severe and cranial nerve palsies are not so apparent If the patient is comatose, other causes of coma such as diabetes mellitus and uræmia must be excluded The diagnosis of meningitis is established by the results of lumbar puncture which also differentiate other varieties of meningitis In tuberculous meningitis a fall in the sugar in the cerebro spinal fluid below mg 45 per 100 ml is an earlier sign than is a fall in the chlorides

*Course and Complications* Tuberculous meningitis is usually rapidly progressive unless adequately treated but a temporary remission with return of consciousness may occur in some cases

**Prognosis** Before the introduction of streptomycin the disease was almost invariably fatal death occurring in 1 to 3 weeks Some cases were more chronic the patient surviving for 2 or 3 months With streptomycin and isoniazid the prognosis is better about 90% of patients recovering if treated early If the patient is comatose if choroidal tubercles are present if the X ray film of the lungs shows a snow storm appearance, or if there is spinal or tentorial block the outlook is usually hopeless

**Treatment** The patient should be on a water bed Treatment must be prolonged for at least a year The tendency now is not to give streptomycin intrathecally It should be injected intramuscularly in doses of 0 5 G daily for a child under 3 years and 1 G daily for a child over 3 years For an adult the dose is 1 G b i d For a child the dose should be continued for 1 to 3 months it is then reduced to 1 G twice a week for 6 months after the cell count and glucose content of the cerebro spinal fluid are normal For an adult the dose is lowered after 6 months to 1 G twice a week for 6 months If vertigo or impairment of hearing occurs the dose must be reduced by 50%

Isoniazid must be given simultaneously by mouth the dose being 8 to 10 mg /kg body weight for a child or adult divided into two doses at 12 hour intervals for 6 weeks The dose is then halved and given twice daily for 12 to 18 months Sedatives such as pot brom gr 5 to 10 t d s for a child of 2 years should be given by mouth and injections of morphin sulph gr $\frac{1}{8}$ may be required The diet should be fluid such as milk, or milk and tea beef tea and barley water Custards and jellies may also be given If the patient cannot swallow he should be fed through a nasal catheter

involvement 3 Mild or ambulatory Characterised by short but severe occipital headache slight pyrexia and perhaps labial herpes 4 Posterior basic meningitis of infants This may be epidemic or sporadic The onset may be insidious but is often sudden with convulsions and vomiting followed by head retraction with opisthotonus The disease soon passes into the chronic stage with wasting hydrocephalus and blindness due to cortical lesions The hands and feet may show the characteristic attitude of tetany Lumbar puncture usually is 'dry' owing to blocking of the foramina of Magendie and Luschka Puncture of the ventricles shows turbid fluid containing meningococci The disease is usually fatal unless adequately treated in 1 or 2 months but recovery may occur the child being often mentally deficient or blind

**Differential Diagnosis** Cerebro spinal fever must be diagnosed from other causes of convulsions in infants such as rickets gastro intestinal disturbances or the onset of the infectious fevers

If there are no meningeal symptoms other causes of continuous temperature must be excluded such as typhoid fever influenza or pneumonia The presence of a purpuric rash and arthritic symptoms may suggest purpura rheumatica and a sudden onset with rash may be mistaken for typhus fever When meningeal symptoms are present it must be diagnosed from meningism other varieties of meningitis polioencephalitis encephalitis lethargica and cerebral tumour Other causes of coma must also be excluded Examination of the cerebrospinal fluid establishes the diagnosis

**Course and Complications** Recrudescence of symptoms during the febrile period which was formerly common is now very rare if adequate treatment is given Relapses after an apyrexial interval are rare

Complications include blindness which may be central or due to suppuration in the eye nerve deafness and hydrocephalus hemiplegia monoplegia paraplegia spastic ataxia bronchopneumonia and arthritis with or without effusion Acute hæmorrhagic nephritis may be the initial symptom Sequelæ include headaches mental instability and pains in the back

*Prognosis* This has been much improved by chemotherapy the mortality being about 5% or lower

**Treatment** *Prophylactic* In an epidemic especially amongst troops the beds should be spaced out to at least 36 inches Tents should not be overcrowded and adequate ventilation ensured A single dose of 2 G of sulphadiazine is said to rid carriers of infection in 24 hours

*Isolation Period* The patient should be isolated until he has recovered and the swab from the naso pharynx shows no meningococci

*Curative* The patient should be put to bed in a well ventilated room The skin is tepid sponged twice a day and all pressure points protected by air rings A water bed may be necessary Diet Nasal feeding may be required but the patient can usually swallow fluids The bowels should be kept open daily with laxatives such as cascara sagrada Enemata may be necessary The abdomen should always be examined to see if there is retention of urine and if so a catheter used

zinski's neck sign (passive flexion of the head results in flexion of the hips and knees) Brudzinski s identical contralateral reflex is less often obtained, when one leg is flexed passively the other follows suit The deep reflexes vary and the plantar reflex may be absent or extensor Cranial nerve palsy is rarely seen but the nerves most likely to be affected are the VI, VIII III and VII in this order of frequency In *infants there is often some bulging of the fontanelle* There may be delirium and the headache may persist throughout Increased drowsiness is generally accompanied by incontinence of urine and fæces The temperature usually remains *raised and is irregularly remittent* The pulse varies with the temperature and is not typically slow Respirations are increased and often irregular Herpes may appear on the lips or on other parts of the body The spleen is sometimes palpable Rapid wasting occurs in some cases during the first week of the disease the subcutaneous tissues being markedly dehydrated

The blood There is a polymorphonuclear leucocytosis of 10,000 per c mm or over The causative organism can frequently be isolated from the blood The urine This rarely shows albuminuria but glycosuria may be present The cerebro spinal fluid This is usually clear for the *first 24 hours and then becomes turbid* and under increased pressure *It* contains pus cells and meningococci (Gram negative intra and extra cellular diplococci) Glucose is usually absent, the chlorides are reduced and the protein is increased

The patient may die within 24 hours or after a few days or the acute stage may be followed about a week later by convalescence the headache and rigidity passing off and the temperature falling to normal or there may be a more prolonged chronic stage

*The Chronic Stage* The chief clinical features are marked wasting despite a good appetite vomiting rigidity muscular twitchings hydrocephalus, dementia convulsions strabismus nerve deafness ptosis and at times facial paralysis There is often incontinence of urine and fæces The cerebro spinal fluid at this stage may show the characteristics of *Froin s syndrome if spinal* block develops above The fluid obtained by lumbar puncture below the block is clear and faintly yellow (xanthochromia) Such a stage may last for 2 to 3 months and the patient may then die or gradually recover

*Varieties* 1 Chronic meningococcal septicæmia without meningitis The chief clinical features are recurrent attacks of fever rigors and small crops of petechiæ seen especially on the lower parts of the *legs the dorsum of the feet and the hands* *The condition can be* diagnosed by blood culture or by finding meningococci in the petechiæ 2 Fulminating Death may occur in a few hours or days the mind remaining clear in the suprarenal type and the patient being stuporose and later comatose in the encephalitic type Cases with hæmorrhage into the suprarenal are known as the Waterhouse Friderichsen syndrome The onset of the disease is sudden with pallor and cyanosis which may be heliotrope and resemble that of influenza Cutaneous hæmorrhages rapid feeble pulse low blood pressure rapid and shallow breathing are characteristic features The low blood pressure indicates the suprarenal

**Differential Diagnosis** The presence of meningitis and its type is determined by the results of lumbar puncture

**Course and Complications** The disease is rapidly progressive

**Prognosis** Septic meningitis was almost always fatal but the prognosis has been much improved by chemotherapy and antibiotics

**Treatment** The general treatment is as described above for tuberculous and meningococcal meningitis For streptococcal staphylo coccal and gonococcal meningitis sulphadiazine or sulphadimidine and penicillin should be given as for meningococcal meningitis The penicillin should first be given intrathecally Crystalline sodium penicillin G made up in pyrogen free saline 10 000 units per 5 ml is used Five ml are injected after removing 5 ml of cerebro spinal fluid At the same time 1 million units of crystalline penicillin are injected intramuscularly and repeated every 8 hours It is advisable to test the sensitivity of the organisms in the cerebro spinal fluid Thus if staphylococci are found to be penicillin resistant streptomycin or aureomycin may be the drug of choice H influenzæ coliform and streptococcus fæcalis infections are usually best treated with strepto mycin mg 50 to 100 being injected intrathecally daily and 1 G intramuscularly every 12 hours The treatment should be continued for a week after the fluid has become clear If streptomycin fails sulphadiazine and penicillin should be given Streptococcal meningitis is often associated with intracranial abscess and ventricular puncture or ventriculography should be performed to exclude this as soon as the streptococci are found in the fluid

## Pneumococcal Meningitis

**Etiology** The Diplococcus pneumoniæ (pneumococcus) is the causative organism A primary form is described in which the meninges are first affected but meningitis is usually secondary to pneumonia empyema or pericarditis the organisms being carried in the blood stream Direct spread may occur from a pneumococcal otitis media

**Clinical Findings** The clinical picture closely resembles that of septic meningitis (see p 310) There is headache vomiting constipation fever neck rigidity and often convulsions with a terminal stage of coma The cerebro spinal fluid This is turbid and under increased pressure An excess of leucocytes is present with pneumococci The protein is increased and the sugar content is reduced

**Differential Diagnosis** The diagnosis is established by the results of lumbar puncture

**Course and Complications** The disease is usually quickly progressive unless relieved by treatment

**Prognosis** The prognosis has been much improved by sulphonamide and penicillin treatment

**Treatment** Sulphadimidine and penicillin should be given The initial dose of sulphonamide should be high and the subsequent doses sufficient to maintain a concentration of 10 to 15 mg per 100 ml in the cerebro spinal fluid Very young infants are given 1 to 3 G and older children and adults 6 to 12 G by mouth as an initial dose This

Drugs For the headache insomnia and pains aspirin gr 5 to 10 t d s bromides gr 10 t d s paraldehyde m 120 to 240 at night or an injection of morphin sulph gr ¼ or hyoscin hydrobrom gr 1/200 may be necessary Ice may be applied to the shaved head and hot baths used to ease the pain in the back

Lumbar puncture relieves headache and tends to prevent hydro cephalus It is required for 2 to 3 days in a few cases to relieve pressure symptoms Frequently it *is only necessary at the beginning* and end of the treatment in order to establish the diagnosis and to ascertain that the fluid has returned to normal Soluble sodium sulphadiazine is first injected intravenously The dose is 0 05 to 0 1 G of sodium sulphadiazine per kg body weight It is administered as a 0 5% solution in physiological saline in order to achieve a blood concentration of 10 to 12 mg /100 ml This is given for 2 days Subsequently the same daily dose of sulphadimidine or of sulphadiazine is given divided into 4 doses administered every 6 hours for about a week An adult should drink 4 to 5 pints of fluid every 24 hours and the urine should be kept alkaline with a potassium citrate mixture administered 3 or 4 times a day

Penicillin is not nearly as efficacious as the sulphonamides, and must be given in high dosage 1 to 2 million units intramuscularly every 2 hours for an adult and half the dose for a child Should symptoms of circulatory collapse be evident the treatment for an Addisonian crisis (see p 701) is required

### Septic Meningitis

*(Pyogenic or Suppurative Meningitis)*

**Etiology** Septic meningitis may be due to infection with the streptococcus staphylococcus the Neisseria gonorrhœæ (gonococcus) the Hæmophilus influenzæ or coliform organisms such as the Bacterium alkaligenes It may follow a direct spread of infection from otitis media *cranial sinusitis mastoiditis or a cerebral abscess In other cases it is* secondary to septicæmia

**Pathology** A purulent infiltration of the meninges covers the vertex and less often the base of the brain The ventricles of the brain may be distended

**Clinical Findings** The symptoms and signs resemble those described above for other forms of meningitis such as tuberculous or meningococcal *Thus there is headache vomiting* and usually constipation

*On Examination* The temperature is raised to 101 F or higher the pulse being about 90 or 100 and often irregular owing to prema ture systoles There is irritability and rigidity of the neck and back muscles There may also be strabismus and unequal pupils The patient is often delirious and rigors may occur The blood There is a leucocytosis of about 12 000 to 20 000 per c mm The cerebro spinal fluid This is turbid and under increased tension There is an excess of leucocytes and the causative organism is present The protein is increased and the sugar content is low

**Differential Diagnosis** The diagnosis is established by the course of the disease the examination of the cerebro spinal fluid and agglutina tion tests on the serum The Paul Bunnell test is of value in the diagnosis of glandular fever It is very liable to be mistaken for tuberculous meningitis until the cerebro spinal fluid has been examined Leptospiral meningitis may closely resemble clinically acute aseptic meningitis In the former there is often ciliary congestion and the diagnosis is confirmed by the agglutination test

**Course and Complications** The disease is of short duration and there are no complications

**Prognosis** The patient usually recovers in about 7 to 10 days

**Treatment** No special treatment is required beyond a lumbar puncture to establish the diagnosis or relieve tension

### Meningism

This is a condition in which there are signs and symptoms of meningeal irritation but the cerebro-spinal fluid is normal and is not under increased tension It is probably due to the presence of toxins in the cerebro spinal fluid It may occur in apical pneumonia especially in children or in acute infections

### Arachnoiditis

*(Meningitis Serosa Circumscripta)*

Arachnoiditis may result from trauma or infection or it may be associated with diseases such as disseminated sclerosis tabes dorsalis syringomyelia and cerebral tumours or it may occur as a sequela of leptomeningitis Cerebral arachnoiditis is often situated in the region of the cortex the optic chiasma or in the posterior fossa The symptoms and signs resemble those of cerebral tumour as the arachnoiditis is characterised by cyst formation Primary optic atrophy may result from chiasmal arachnoiditis *Otitic hydrocephalus* occurring in associa tion with middle ear infection is closely allied to arachnoiditis It is suggested that infection of the lateral sinus leads to retrograde throm bosis of the superior longitudinal sinus Absorption of the cerebro spinal fluid through the arachnoid villi is thereby blocked and hydro cephalus results Treatment consists in repeated lumbar puncture Spinal arachnoiditis gives rise to root pains followed by varying degrees of paraplegia At operation a gush of fluid escapes from the arachnoid cyst the arachnoid itself showing very slight signs of inflammation

## THE CEREBRUM

### Intracranial Aneurysms

**Pathology** The following varieties are described and the order of frequency of their occurrence is 1 *Congenital* These are thought to be due to weak spots in the muscle coat and are met with especially on the base of the brain at the junction of the anterior cerebral and anterior communicating arteries They are said to resemble a berry hanging from a stalk From the clinical standpoint these are of the greatest

is followed by a quarter of the initial dose every six hours until the patient seems entirely well clinically This dose should not be reduced until the temperature has been normal for a week, and half the amount is then given for several days until two successive cultures of the cerebro spinal fluid are sterile Penicillin should be given as follows —10 000 units of crystalline sodium penicillin G dissolved in 5 ml of pyrogen free saline are injected intrathecally daily, after removal of 5 ml of cerebro spinal fluid for about 5 days and 1 million units of penicillin are given intramuscularly every 4 hours for 5 days

If the cerebro spinal pathways become blocked by fibrino purulent material as shown by spinal block on lumbar puncture, 5,000 to 10 000 units of penicillin should be injected into the lateral ventricles, through burr holes in the skull daily for 4 to 7 days Sulphadimidine should also be given by mouth or nasal tube 4 G followed by 2 G every 4 hours for 5 days

### Acute Lymphocytic Chorio Meningitis

(*Acute Aseptic Meningitis Acute Serous Meningitis Acute Benign Lymphocytic Meningitis*)

**Definition** A meningitis of acute onset with a favourable course, in which the cerebro spinal fluid is sterile

**Etiology** About one third of the cases of acute lymphocytic meningitis are due to three viruses one found in mice one causing swineherd s disease in France Switzerland and Italy, and the third producing pseudo lymphocytic chorio meningitis in England The virus has also been found in the spleen of dogs A monocytic meningitis may occasionally occur in glandular fever, infective jaundice and mumps Acute idiopathic benign serous meningitis may complicate otitis media cerebral abscess *chronic alcoholism* (" *wet brain* ) *or encephalitis* lethargica

**Clinical Findings** The patient may be a child or adult He is suddenly taken ill with headache (usually occipital) vomiting stiffness of the neck pains in the neck and limbs and insomnia The mind remains clear Lymphocytic chorio meningitis often has an influenza like onset the meningeal symptoms not appearing for 2 to 8 weeks

*On Examination* There is some rigidity of the neck and back, and Kernig s and Brudzinski s signs are present There may be nystagmus and a squint The temperature is raised to about 100° or 101° F, and the pulse *is about 80 to 90 Ophthalmoscopic examination* often reveals swelling of the disc (papilloedema) At times a transient facial palsy may be observed The cerebro spinal fluid This is under increased pressure clear or slightly turbid There is an increase of cells, usually of lymphocytes In some cases the polymorphonuclear cells are first increased and later in the illness there is an excess of lymphocytes The protein may be increased but the sugar and chloride content are normal No organisms are present but in certain *cases the virus can* be isolated by animal inoculation during the first week of the meningitis the result not being available however for 2 to 8 weeks



FIG 23 CEREBRAL ARTERIOGRAM SHOWING ANEURYSM OF THE ANTERIOR COMMUNICATING ARTERY

importance 2 *Arteriosclerotic* Degeneration occurs in the media 3 *Infective embolic or mycotic* These result from adhesion of an infected embolus to the intima, and may occur in infective endocarditis or septicæmia There is a tendency to rapid formation and rupture Mycotic aneurysms are often found on the middle cerebral artery and occur chiefly in young people 4 *Acute polyarteritis nodosa* This is another cause, the outer coat of the artery being first affected 5 *Syphilitic* The basilar artery is chiefly affected Some authorities state that this kind of aneurysm does not occur 6 *Traumatic* This is usually of the arterio venous type, and situated between the internal carotid artery and the cavernous sinus

The aneurysms vary in size from that of a pin s head to a fist the average size being that of a pea They are usually situated near the base of the brain but may be deep in a hemisphere The middle cerebral and the basilar arteries are those most commonly affected Rupture occurs in about 50% of all cases

**Clinical Findings** *Before rupture* There are often no signs or symptoms before rupture occurs but in some cases there is headache, usually homolateral irritability giddiness tinnitus and nausea Papillœdema may be present and a localised systolic murmur is some times heard on listening to the skull over the aneurysm, especially in the arterio venous type Variability of signs from time to time is a feature of unruptured intracranial aneurysms Other localising signs may be present which vary with the artery affected *Internal carotid* This results in pressure on the III IV and VI and upper division of V nerves, with partial or complete internal and external ophthalmoplegia and pain or sensory loss over the forehead and eye Pressure further forward on the optic path may result in hemianopia and optic atrophy Non pulsating exophthalmos may also occur *Middle cerebral* There may be Jacksonian fits and hemiplegia or monoplegia *Anterior cerebral and anterior communicating* (See Fig 25) The signs may resemble those of a frontal lobe tumour *Vertebral* The syndrome pro duced may resemble that of thrombosis of the posterior inferior cerebellar artery *Basilar* The III V VI, VII or VIII nerves may be affected or there may be pressure on the pituitary *Arterio venous aneurysm* of the internal carotid artery and cavernous sinus This is a cause of pulsating exophthalmos

*At rupture* When rupture occurs the symptoms are usually those of subarachnoid hæmorrhage but at times the hæmorrhage is intracerebral

**Differential Diagnosis** Intracranial aneurysms should be thought of when there are symptoms of a cerebral tumour or of involvement of the III IV V and VI nerves When rupture occurs and blood is found in the cerebro spinal fluid other causes of cerebral hæmorrhage have to be considered such as hæmorrhage into a glioma The skull should be X rayed for in some instances the calcified aneurysm is revealed The shadow may be as large as a walnut Albl describes an almost closed circle shadow but only a portion of the arc or several arcs of circles may be shown Angiography will usually reveal the exact site of the aneurysm

**Prognosis** Death occurs in about 60% of the cases which bleed

**Treatment** When facilities are available angiography should be performed as soon as possible to locate the site of the aneurysm It must then be decided whether an attempt should be made to deal surgically with the aneurysm or to ligate the carotid artery on the affected side Rest in bed is essential and in some cases a sleepy state may be induced for periods up to 2 months by the administration of hypnotics such as phenobarbitone in adequate doses Thus pheno barbitone gr 1 t d s may be required Lumbar puncture may be advisable (see Subarachnoid hæmorrhage p 318) If the blood Wasser mann reaction is positive anti syphilitic treatment should be given (see p 596)

## Intracranial Hæmorrhage

**Definition** Hæmorrhage into the brain or meningeal spaces

**Anatomy** Blood is brought to the brain by the two vertebral and the two internal carotid arteries The vertebral arteries pass along the ventral surface of the medulla and unite at the lower border of the pons to form the basilar artery At the upper border of the pons the basilar artery divides into the two posterior cerebral arteries These are connected with the anterior cerebral arteries by the posterior communicating arteries and in this way there is formed the circle of Willis lying in the interpeduncular space on the base of the brain The internal carotid arteries give off the anterior cerebral arteries and are then continued as the middle cerebral arteries The anterior cerebral arteries are connected by the anterior communicating artery (see Fig 26) The anterior cerebral artery supplies the first frontal convolution part of the second frontal and the upper part of the ascending frontal and parietal convolutions The middle cere bral artery supplies the motor and sensory areas of the cerebral cortex including the speech area The posterior cerebral artery is distributed to the ventro mesial portions of the temporal and occipital lobes Central branches enter the brain substance from the anterior middle and posterior cerebral arteries and from the circle of Willis The lenticulo striate branch of the middle cerebral artery is distributed to the external capsule the lenticular nucleus the anterior part of the internal capsule and the caudate nucleus It is a frequent site for a cerebral hæmorrhage The lenticulo-optic branch supplies the posterior part of the internal capsule The anatomy of the cranial venous sinuses is given on p 324

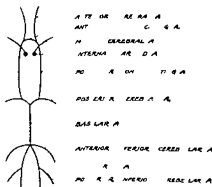


Fig 26 Diagram of the Circle of Willis

The chief types of intracranial hæmorrhage will now be described

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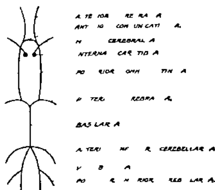


Fig 26 Diagram of the Circle of Willis

The chief types of intracranial hæmorrhage will now be described

### Extradural or Epidural Hæmorrhage

**Etiology** This is associated with concussion and injury to the skull and is also met with in hæmorrhagic pachymeningitis It is most often due to a tear in the middle meningeal artery caused by fracture of the skull

**Clinical Findings** At the onset the patient is generally unconscious and convulsions may occur according to the part of the brain stimulated by the hæmorrhage He may then recover consciousness for a few hours and appear much better Recovery may then take place often associated with a post concussional state or this lucid interval " may be followed by coma and death

**Treatment** X ray of the skull, angiography and electro encephalo graphy are all of value in localising the site of the bleeding When the hæmorrhage is due to trauma and there is evidence that the bleeding is continuing the skull should be trephined and an endeavour made to ligature the bleeding vessel and evacuate the clot

### Concussion and the Post Concussional State

**Pathology** The nature of the concussional cerebral changes due to head injuries remains obscure despite much experimental investiga tion Perivascular petechial hæmorrhages resulting from diapedesis may be found usually in the cortex and often in the subjacent white matter and in the grey matter of the brain and brain stem

**Clinical Findings** After a minor injury the patient may be tempo *rarily unconscious exhibiting a flaccid paralysis The pulse is feeble* temperature subnormal, respirations shallow and slow and the pupils are dilated Recovery of consciousness may be rapid or gradual The patient may then be unaware of the accident or he may remember the accident and what happened for a short time after it He may now pass into an automatic state during which he performs actions of which he has no subsequent recollection The boxer who fights while 'punch drunk' affords an example of *automatism After a* severe injury the patient immediately loses consciousness and days or even months may elapse before it is fully restored Certain stages are described in the process of recovery These may overlap and all may not be present The initial coma and flaccid paralysis are followed by restlessness and stupor which persist for a few days This may pass into a stage of excitement bewilderment and delirium in which the patient cannot feed himself and is incontinent *This stage may last for several* weeks or months and may be interrupted by short periods of stupor About this time the patient can usually talk more rationally and answer questions The characteristics of this traumatic psychosis are a dis orientation in time and space retrograde amnesia speech disturbances confabulation and perseveration Later there is a marked defect of memory for recent events (Korsakow s psychosis) Traumatic hysteria is very liable to follow head *injuries Residues which may persist for* some time include headache difficulty in concentration abnormal fatiguability forgetfulness and intolerance of even small amounts of alcohol

### Acute Subdural Hæmatoma

**Etiology** This results from the rupture of veins running across the subdural space perhaps from the result of a slight blow on the head

**Clinical Findings** The symptoms occur within a few hours of the injury Sudden attacks of coma with evidence of upper motor neurone lesions followed by equally sudden return to consciousness and disappearance of physical signs are very suggestive Death is likely to occur unless a bilateral subtemporal trephining is performed

### Chronic Subdural Hæmatoma

**Etiology** The most common cause is trauma and it may result from birth injury Less often it results from purpura scurvy carcinoma of the dura neurosyphilis or chronic alcoholism

**Clinical Findings** After a latent period of a few weeks or months following a blow the patient complains of intermittent headache and mental dulness gradually increasing in severity Later cerebral irritation may ensue with convulsions or hemiparesis Papillœdema is not constantly present The cerebro spinal fluid may be under increased pressure and contain blood The combination of signs of increased intracranial pressure with a normal pressure of cerebro spinal fluid is very suggestive of a subdural hæmatoma The hæmatoma is often situated in the parietal region and may be bilateral Localisation is aided by angiography

**Treatment** The clot should be removed surgically It is usually advisable to explore both sides of the brain

### Subarachnoid Hæmorrhage

**Etiology** Subarachnoid hæmorrhage is usually due to rupture of a congenital or mycotic cerebral aneurysm Other causes include head injuries effusion of blood from an intracerebral hæmorrhage a leak from an angioma hæmorrhagic encephalitis or blood diseases

**Clinical Findings** A congenital intracranial aneurysm usually causes no symptoms until it begins to leak Slight degrees of bleeding may give rise to headache In more severe cases the patient experiences intense headache often first occipital and then frontal he then vomits and rapidly loses consciousness

*On Examination* The patient is semi conscious and looks very ill The pulse is frequent breathing stertorous and the temperature may rise to 101 F or higher The abdominal reflexes and tendon reflexes are usually depressed or absent and an extensor plantar response may be present on one or both sides There may be a certain amount of neck rigidity or head retraction and convulsive movements of the extremities The pupils are dilated and papillœdema retinal or subhyaloid hæmorrhages the latter being situated between the vitreous and the retina may be seen on ophthalmoscopic examination The urine may contain a considerable amount of albumin for a few days Glycosuria is present at times with traces of acetone If a lumbar puncture is performed, the fluid is found to be uniformly mixed with

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**Clinical Findings** At the onset the patient is generally unconscious and convulsions may occur according to the part of the brain stimulated by the hæmorrhage He may then recover consciousness for a few hours and appear much better Recovery may then take place, often associated with a post concussional state *or thus " lucid interval " may be followed* by coma and death

**Treatment** X ray of the skull angiography and electro encephalography are all of value in localising the site of the bleeding When the hæmorrhage is due to trauma and there is evidence that the bleeding is continuing the skull should be trephined and an endeavour made to ligature the bleeding vessel and evacuate the clot

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**Clinical Findings** There may be a history of previous slight attacks or "little strokes" from which the patient has recovered to a varying degree These are usually due to cerebral thromboses The onset is generally sudden the patient falling unconscious with the apoplectic stroke Less often there are prodromal symptoms such as headache giddiness vomiting disturbance of speech or tingling and weakness in a limb The results produced vary with the site of the lesion

1 *Capsular Hæmorrhage* This is the most common variety, the bleeding starting external to the internal capsule

*On Examination* The patient is unconscious the face is usually flushed cyanosed and sweating rarely it is pale The breathing is stertorous and the cheek on the paralysed side may be blown in and out with respiration There is no movement of the limbs which are flaccid On lifting up an arm or leg a greater degree of flaccidity may be detected on the paralysed side In the early irritative stage there may be conjugate deviation of the eyes which look away from the site of the lesion in the brain later with a paralytic lesion the deviation is in the opposite direction towards the site of the lesion The corneal superficial and deep reflexes are lost There may be incontinence or retention of urine and incontinence of fæces The pulse is full and bounding and may be slow or rapid The temperature is usually subnormal at the onset and the blood pressure is raised The urine often contains albumin and sugar may be present The cerebro spinal fluid contains blood if the hæmorrhage has extended into the lateral ventricle If the patient survives he will pass in a few days into a post apoplectic stage described on p 322 under the title ' chronic residual hemiplegia

2 *Cortical Hæmorrhage* This is rare The patient does not generally lose consciousness Convulsions or paralysis of one or more limbs aphasia or hemianopia may develop according to the site of the lesion

3 *Thalamic Hæmorrhage* This will produce the thalamic syndrome (see p 329)

4 *Mid brain Hæmorrhage* There may be paralysis of the III nerve with hemiplegia on the opposite side (Weber s syndrome) There may also be anæsthesia of the paralysed side of the body

5 *Pontine Hæmorrhage* The patient rapidly becomes comatose There may be convulsions of the legs with vomiting Cheyne Stokes breathing and pin point pupils Conjugate deviation of the eyes may also be noted in a direction opposite to that characteristic of a supra nuclear lesion (see p 383) There may be contralateral hemiplegia The pupils may dilate before death The temperature rises to 106 F or higher and the patient dies within a few hours

6 *Medullary Hæmorrhage* The patient usually dies in coma within a few hours before signs of bulbar paralysis are apparent

7 *Ventricular Hæmorrhage* This is usually secondary to capsular hæmorrhage causing rapid death There may be head retraction with rigidity or spasms of the arms or legs

8 *Cerebellar Hæmorrhage* (See p 371 )

blood which does not clot on standing Red cells disappear from the fluid about 7 days after the hæmorrhage has ceased The bleeding may stop and the patient recover, having various sequelæ, such as headache disturbance of vision and of mentality, or certain paralyses The III V, VI or VII nerves may be pressed on with partial III nerve palsy, intense pain on one side of the face, abducens palsy, or lower motor neurone facial palsy In other cases the hæmorrhage continues generalised bronchitis and œdema of the lungs develop and the patient dies in the course of a day or so *The anterior choroidal syndrome* may quickly follow a subarachnoid hæmorrhage This is characterised by contralateral hemiplegia hemianæsthesia and hemianopia Localisation of the aneurysm may be effected by angiography

**Prognosis** Over 60% of cases of subarachnoid hæmorrhage are fatal

**Treatment** An initial lumbar puncture is advisable to establish the diagnosis but it is usually wise not to repeat the lumbar puncture as a routine measure as this may cause the bleeding to recur It should however be repeated if the coma deepens or the blood pressure rises and if there are convulsions or headache which is not relieved by morphine If there is pulmonary œdema atropin sulph gr 1/100 and morphin sulph gr ¼ should be injected subcutaneously It may be possible to arrest the bleeding by operation

## Intracerebral Hæmorrhage

**Definition** Hæmorrhage into the substance or ventricular system of the brain

**Etiology** Hæmorrhage results from rupture of a cerebral artery or aneurysm or of capillaries cortical cerebral veins or venous sinuses Exciting causes include muscular strain and trauma to the skull *Predisposing causes* 1 Age Usually over 40 years In children hæmorrhage may occur from rupture of cortical veins in whooping cough there may be hæmorrhage into a glioma or rupture of an intracranial aneurysm in infants hæmorrhage may be due to birth injuries or rupture of an intracranial aneurysm 2 Sex Males predominate 3 Heredity Cerebral hæmorrhage tends to run in families 4 *Arteriosclerosis angiospasm high blood pressure and chronic nephritis* 5 Blood diseases such as leukæmia or purpura

**Pathology** The lenticular branches of the middle cerebral artery are most frequently affected the lesion being situated *commonly in* the thalamus, internal capsule and corpus striatum Spread to the ventricular system is frequent In blood diseases hæmorrhage may occur by diapedesis *In all cases of cerebral hæmorrhage* bleeding usually continues until the patient dies Congenital aneurysms may however leak from time to time before the fatal hæmorrhage occurs *Further an old blood cyst may* be found in the brain at autopsy which indicates the site of a previous hæmorrhage from which the patient has recovered Meningeal hæmorrhage due to birth injury may lead *to* atrophy or to the formation of cysts in the brain (porencephaly) The pathology of intracranial aneurysms is considered on p 318

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**Etiology** Hæmorrhage results from rupture of a cerebral artery or aneurysm or of capillaries cortical cerebral veins or venous sinuses Exciting causes include muscular strain and trauma to the skull *Predisposing causes* 1 Age Usually over 40 years In children hæmorrhage may occur from rupture of cortical veins in whooping cough there may be hæmorrhage into a glioma or rupture of an intracranial aneurysm in infants hæmorrhage may be due to birth injuries or rupture of an intracranial aneurysm 2 Sex Males predominate 3 Heredity Cerebral hæmorrhage tends to run in families 4 Arteriosclerosis angiospasm high blood pressure and chronic nephritis 5 Blood diseases such as leukæmia or purpura

**Pathology** The lenticular branches of the middle cerebral artery are most frequently affected the lesion being situated commonly in the thalamus internal capsule and corpus striatum Spread to the ventricular system is frequent In blood diseases hæmorrhage may occur by diapedesis In all cases of cerebral hæmorrhage bleeding usually continues until the patient dies Congenital aneurysms may however leak from time to time before the fatal hæmorrhage occurs Further an old blood cyst may be found in the brain at autopsy which indicates the site of a previous hæmorrhage from which the patient has recovered Meningeal hæmorrhage due to birth injury may lead to atrophy or to the formation of cysts in the brain (porencephaly) The pathology of intracranial aneurysms is considered on p 313

present the diagnosis of hæmorrhage is confirmed if it is absent stimulants such as Coramine 1 5 ml should be administered sub cutaneously six hourly If the patient survives nasal feeding is often required for a time and penicillin should be given to prevent basal bronchial infection Catheterisation may be necessary In some cases it is possible to locate the site of the bleeding by angiography and to remove surgically the clot with good results

### Cerebral Thrombosis

**Definition** Coagulation of blood in the cerebral vessels

**Etiology** *Predisposing causes* Arteriosclerosis syphilitic endarteritis low blood pressure chronic nephritis slowing of the circula tion in various debilitating conditions polycythæmia rubra anæmia septicæmia encephalitis lethargica and polioencephalitis Cerebral thrombosis may also result from trauma such as a wound of the brain or develop in the neighbourhood of a cerebral tumour or be secondary to a cerebral embolus Adults over middle age are usually affected syphilis being the commonest cause in young adults

**Pathology** The middle cerebral artery is most frequently affected The resultant infarct may be red or white The central arteries are end arteries and so softening of the brain with degeneration of nerve elements usually results from their obstruction and later a scar or cyst may form The anastomoses are better developed for the cortical arteries

**Clinical Findings** If thrombosis affects a large artery the onset of symptoms is acute with coma When smaller vessels are involved there are often prodromal symptoms Thus the patient may complain for a few days of headache giddiness disturbance of speech numbness or tingling of a hand or arm with subsequent paralysis In some cases there are convulsions at the onset

*On Examination* If a large vessel is blocked the patient is comatose and the clinical findings resemble those described for cerebral hæmor rhage The signs depend upon the site of the lesion 1 *Middle cerebral artery thrombosis* There is usually hemiplegia At the onset the affected side of the body is flaccid and the superficial and deep reflexes are abolished The lower half of the face is affected and also the tongue so that on protrusion it deviates towards the paralysed side The muscles of mastication deglutition and the trunk muscles are not usually paralysed There is some weakness in turning the head and eyes to the paralysed side In a day or so the plantar response is extensor on the affected side and the abdominal reflex is absent on the paralysed side but present on the sound side 2 *Anterior cerebral artery thrombosis* There may be no symptoms or dementia may ensue The grasp reflex (see p 328) may be present in one or both feet Contralateral crural monoplegia may rapidly develop 3 *Posterior cerebral artery thrombosis* There may be homonymous hemianopia the patient having difficulty in avoiding objects on his blind side Hemianæsthesia may also be present 4 *Internal carotid*

**Differential Diagnosis** The diagnosis *includes a consideration of* cerebral thrombosis cerebral embolus a hypertensive cerebral attack and other causes of coma In cerebral thrombosis the onset is more *insidious and more likely to occur at night* The patient may experience numbness or tingling in the limb, followed during the course of a few hours by paralysis The cerebro spinal fluid does not contain blood In cerebral embolus the onset is usually sudden but often the patient does not lose consciousness unless an extensive area of brain is involved There may be aphasia, monoplegia hemiplegia or supranuclear paralysis of the VII cranial nerve The age incidence is often lower than in hæmorrhage and usually a cardiac lesion, such as mitral stenosis or auricular fibrillation is present Cerebral thrombo phlebitis secondary to systemic thrombosis elsewhere may closely simulate cerebral embolus The spread of infection is considered to be by mural thrombo phlebitis, *along the intravertebral veins* The lungs are *not often affected and a* mistaken diagnosis of paradoxical embolus (see p 323) may be made Other causes of coma include uræmia, diabetes mellitus cerebro spinal meningitis, opium and alcohol In some cases epilepsy and hysteria will require exclusion In uræmia the urine contains protein and casts and the percentage of urea in the blood and cerebro spinal fluid is generally raised In diabetic coma the breath smells of acetone and the urine contains sugar and acetone bodies In cerebral hæmorrhage although sugar may be present in the urine acetone bodies are absent In hypoglycæmic coma the patient may have convulsions but a history of diabetes with insulin administration can be obtained and consciousness is usually restored by the subcutaneous injection of 1 ml of liq adrenal hydrochlor Examination of the cerebro spinal fluid enables a diagnosis of cerebro spinal fever to be made In opium poisoning the pupils are pin point, and some evidence of the administration of the drug or its derivatives can usually be found Alcohol poisoning may cause difficulty as the patient may have a hæmorrhage while under the influence *of alcohol and so he should always be kept under observation* for 24 hours In epilepsy the unconscious and convulsive phases are generally of comparatively short duration and if there is resultant hemiplegia (*Todd s paralysis*) *recovery is complete* The *nature of a* hysterical attack is usually clear (see p 864)

**Course and Complications** It is doubtful if a patient can survive a cerebral hæmorrhage in some cases after an apparent improvement with recovery of consciousness there is recurrence of coma followed by death due to spread of the bleeding into the lateral ventricle

**Prognosis** A cerebral hæmorrhage is usually fatal in from 2 to 48 hours

**Treatment** The patient should be propped up in bed all tight clothing loosened dentures removed and an airway maintained by turning him slightly on one side or by using an anæsthetist s airway If there is marked venous engorgement in the neck and cyanosis 10 oz of blood should be removed from a vein and the bowels opened by an enema A lumbar puncture is not without danger as it may lead to compression of the medulla in the foramen magnum If blood is

a cervical rib 6 The pulmonary veins in suppuration of the lungs 7 The systemic veins if there is a patent foramen ovale or patent inter ventricular septum (paradoxical embolism) 8 Fat emboli resulting from fracture of long bones may pass through the lungs and lodge in the brain *Predisposing causes* 1 Age Adults and young people 2 Sex Females predominate

**Pathology** The left middle cerebral artery is most often obstruc ted the effects produced resembling those described for cerebral thrombosis

**Clinical Findings** The onset is sudden and there are no prodromal symptoms There may be convulsions aphasia monoplegia hemiplegia visual disturbances or facial palsy of the upper neurone type according to the part of the brain affected Consciousness may or may not be lost depending upon the extent of brain involved Carotid hemiplegia is due to an embolus obstructing the internal carotid artery there is homolateral blindness and heterolateral hemiplegia In many cases an associated cardiac lesion is present and in some instances the embolus forms during the administration of quinidine

**Differential Diagnosis** This is as described for cerebral hæmorrhage

**Course and Complications** In carotid hemiplegia there is often restoration of function due to establishment of a collateral circulation through the circle of Willis Recurrent emboli are liable to form In the majority of cases the course of the lesion resembles that described for cerebral thrombosis

**Prognosis** There is usually recovery of the use of the leg in hemiplegia but the hand and arm often remain permanently paralysed and speech may be deranged especially when the patient is excited Death results if a large area of the brain is deprived of its blood supply

**Treatment** Nicotinic acid mg 100 should be injected intravenously to dilate the cerebral arteries with the hope that the embolus will pass to a smaller vessel Apart from this treatment is as described for cerebral thrombosis

## Sinus Thrombosis

**Definition** Coagulation of blood in the cranial venous sinuses

**Etiology** 1 *Primary sinus thrombosis* This is an aseptic process In infants it may be due to marasmus or to congenital syphilis and in adults to anæmia and to wasting diseases such as tuberculosis or carcinoma The superior longitudinal sinus is usually affected 2 *Secondary sinus thrombosis* This results from sepsis as (*a*) in the middle ear (lateral sinus thrombosis) (*b*) in the cranial air sinuses (*c*) in the skull due to caries or a fracture (*d*) in the orbit nose or upper lip Thus it may follow a boil carbuncle or insect bite (cavernous sinus thrombosis) In other cases the infection spreads from the ear mouth or throat It may also be a manifestation of thrombo phlebitis migrans or be secondary to peripheral systemic thrombosis which has spread to the brain along the intravertebral veins

**Anatomy and Pathology** The anatomy of the important cranial

*thrombosis* This causes coma and rapid death 5 *Ba[silar artery]* *thrombosis* This produces symptoms resembling those de[scribed under] *pontine hæmorrhage* 6 *Vertebral artery thrombosis* This m[ay produce] symptoms of acute bulbar paralysis

**Differential Diagnosis** This is as described for cerebral h[æmorrhage] (see p 320)

**Course and Complications** After the acute phase of [the illness] has passed, in cases of middle cerebral artery thrombosis, [the patient] enters the stage of "chronic residual hemiplegia" Recovery [of some] movements is obtained, the arm being always more paralyse[d than the] leg Thus there is power of extension of the hip and knee, and [dorsi-]flexion of the ankle and toes As regards the arm the patien[t cannot] abduct and elevate it, and to flex the elbow, wrist and fin[gers. The] muscles producing these movements become spastic and [the tendon] reflexes are increased due to the uncontrolled activity of [the extra-]pyramidal motor paths Ankle clonus and knee clonus are [often] obtained and the plantar response remains extensor The [abdominal] reflex may return on the paralysed side There are al[so] associated reactions thus if the patient yawns he may [extend his] wrist and fingers and raise his hand in front of his face, [performing] movements involuntarily which he cannot achieve voluntaril[y]

**Prognosis** This is always unfavourable Recurrent thro[mbosis or] a fatal cerebral hæmorrhage is liable to ensue

**Treatment** The patient should be kept in bed at the c[ommencement and] stimulants administered such as subcutaneous injections of [...] 1 5 ml t i d or of strychnin hydrochlorid gr 1/32 t i d Anti[coagulant] treatment should be avoided if the diagnosis from cerebral hæ[morrhage] is uncertain In little strokes anticoagulant treatment (s[ee ...]) is of value The Wassermann reaction should be determin[ed and if] positive crystalline penicillin should be injected intram[uscularly] 1 million units daily for 21 days Splints should be appli[ed to the] affected arm to prevent flexion of the wrist and fingers an[d the leg] should be maintained rotated inwards with the ankle dorsi[flexed by] means of sandbags Gentle massage and passive movements [should be] given in the course of a few days As soon as possible the patie[nt should] be encouraged to perform active movements but electrical sti[mulation] of the muscles is always contra indicated The patient s[hould be] sat in an arm chair as soon as he has recovered from the sho[ck of the] stroke

### *Cerebral Embolus*

**Definition** Obstruction of a cerebral artery by an embolu[s]

**Etiology** The embolus may be derived from the following

1 A valve of the heart, especially the mitral and less freque[ntly the] aortic valve This occurs in chronic endocarditis 2 The lef[t auricle] or ventricle a portion of clot being detached in mitral stenosis [auricular] fibrillation or flutter or in coronary thrombosis 3 The endo[cardium] in malignant or subacute bacterial endocarditis 4 The a[orta in] aneurysm or atheroma 5 The right subclavian artery from pr[...]

a cervical rib 6 The pulmonary veins in suppuration of the lungs 7 The systemic veins if there is a patent foramen ovale or patent inter ventricular septum (paradoxical embolism) 8 Fat emboli resulting from fracture of long bones may pass through the lungs and lodge in the brain *Predisposing causes* 1 Age Adults and young people 2 Sex Females predominate

**Pathology** The left middle cerebral artery is most often obstructed the effects produced resembling those described for cerebral thrombosis

**Clinical Findings** The onset is sudden and there are no prodromal symptoms There may be convulsions aphasia monoplegia hemiplegia visual disturbances or facial palsy of the upper neurone type according to the part of the brain affected Consciousness may or may not be lost depending upon the extent of brain involved Carotid hemiplegia is due to an embolus obstructing the internal carotid artery there is homolateral blindness and heterolateral hemiplegia In many cases an associated cardiac lesion is present and in some instances the embolus forms during the administration of quinidine

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**Treatment** Nicotinic acid mg 100 should be injected intravenously to dilate the cerebral arteries with the hope that the embolus will pass to a smaller vessel Apart from this treatment is as described for cerebral thrombosis

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**Anatomy and Pathology** The anatomy of the important cranial

venous sinuses is indicated in the diagram (see Fig 27) The walls of the sinuses consist of dura mater The blood enters the sinuses from the brain and the meninges The direction of flow in the emissary veins is uncertain The blood passes from all the sinuses into the internal jugular veins There are five single sinuses namely the superior longitudinal the inferior longitudinal the straight the circular and the basilar sinuses There are six paired sinuses namely the lateral the occipital the cavernous the superior petrosal the inferior petrosal, and the spheno parietal sinuses From the medical point of view the most important are the cavernous sinus the lateral sinus and the superior longitudinal sinus The cavernous sinus is in close proximity to the sphenoidal air sinus and only separated from it by a thin layer of bone The internal carotid artery and the VI nerve are on its inner side and the III IV and V nerves pass through its outer wall (see Fig 28) The ophthalmic veins enter it in front and it is also in communication anteriorly with the frontal and supra orbital veins The superior and inferior petrosal sinuses connect it with the lateral sinus behind The circular sinus connects the two cavernous sinuses The superior longitudinal sinus receives blood from the brain and obstruction is liable to cause gross cerebral disturbance Infection probably spreads by means of infected blood clot or along the sheath of the I and VIII nerves There is no lymphatic path At autopsy a clot may be found adhering to the wall of the sinus and in some cases it extends into the jugular and subclavian veins or even into the superior vena cava Organisms such as streptococci or pneumococci may be present in the clot

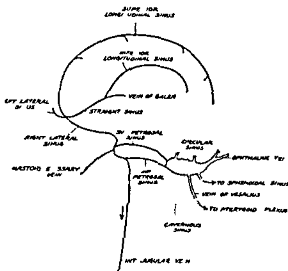



Fig 27 Diagram showing the Cranial Venous Sinuses

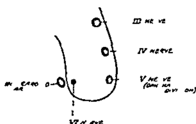



Fig 28 Diagram showing the Cavernous Sinus and its Nerves

**Clinical Findings** *Cavernous Sinus Thrombosis* In a typical case the patient has noticed a sore place or small boil on the upper lip or the inside of the nose It may have been opened surgically A day or so later the patient complains of frontal headache with malaise and

drowsiness Later there may be pain in the side of the face or eye and the vision may fail

*On Examination* At the onset the temperature is found to be raised to about 101° F and the pulse to 90 or 100 Definite signs soon appear and the patient becomes very ill There is protrusion usually of both eyes swelling of the eyelids and œdema of the conjunctivæ and the eyes protrude between the closing eyelids (chemosis) The eyelids themselves are œdematous and may be bluish The swelling may spread to the forehead and side of the face External oculomotor paralysis is commonly found the VI nerve being often first affected and the pupils may be dilated and fixed The infection sometimes travels back to the lateral sinus causing pain behind the ear and some œdema over the mastoid process It may spread even further to the internal jugular vein when there is stiffness pain and swelling of the neck The cerebro spinal fluid This is often under increased pressure and contains an excess of polymorphonuclear cells and organisms

*Lateral Sinus Thrombosis* The patient frequently has been suffering from chronic suppurative otitis media or a mastoid operation may have been performed He complains of headache or vomiting

*On Examination* There may be no otorrhœa the temperature rises and is swinging in type but the pulse is often slow Frequently there are no other signs but papillœdema may be present If the infection spreads to the internal jugular vein the patient will notice difficulty in turning the head to the opposite side and the vein is often felt thrombosed There may also be œdema over the mastoid process

*Superior Longitudinal Sinus Thrombosis* The patient complains of headache vomiting or bleeding from the nose

*On Examination* The anterior fontanelle is seen bulging in infants and there is often enlargement of the veins on the front and side of the head Convulsions and spastic paralysis of both legs may rapidly ensue There may be papillœdema squint or otitic hydrocephalus The patient usually becomes delirious and incontinent The cerebro spinal fluid In infants this is usually clear and under increased pressure with an increase of lymphocytes

**Differential Diagnosis** In cavernous sinus thrombosis the bilateral proptosis with swelling of the eyelids is very characteristic Proptosis associated with retro-orbital tumours is usually afebrile It should always be remembered that with sphenoidal sinusitis in which the cavernous sinus is not affected there may be swelling of the eyelids bulging eyes and headache Transillumination X ray examination and exploratory puncture should serve to differentiate Further cavernous sinus thrombosis may occur without proptosis It is not usually possible to diagnose lateral sinus thrombosis with certainty apart from exploratory operation Examination of the cerebro spinal fluid will indicate whether meningitis is present but meningitis may also occur as a complication of the sinus thrombosis

**Course and Complications** The course is usually rapid complications include meningitis cerebral abscess and pyæmia

**Prognosis** This is very grave, but has been considerably improved by the use of penicillin

**Treatment** *Prophylactic* Septic spots on the upper lip and nose should be fomented and not squeezed, and early surgical intervention is dangerous When pus definitely forms it should be evacuated through a small incision All cases of otorrhœa should be adequately treated

*Curative* Crystalline penicillin should be given, 60 000 units intramuscularly every 8 hours for 7 to 10 days Successful results have been obtained by this treatment In lateral sinus thrombosis it may be advisable to ligature the jugular vein to prevent spread of infection

### Intracranial Tumours

**Definition** New growths granulomata and cysts arising in the brain meninges or interior of the skull

**Etiology** In some cases of intracranial tumour there is a history of a blow on the head, followed by the signs of the tumour These signs may result from the occurrence of œdema or hæmorrhage in or around a pre existent tumour *Predisposing causes* 1 Age The majority of tumours occur in young or middle aged people Glioma and tuberculoma may affect children 2 Sex Males predominate slightly

**Pathology** The following tumours may occur and they are grouped approximately in their order of frequency Glioma (including astrocytoma multiple spongioblastoma medulloblastoma ependymoma etc) pituitary adenoma, meningioma (endothelioma) acoustic neuro fibroma congenital cysts secondary malignant growths such as sarcoma carcinoma and hypernephroma gumma tuberculoma vascular tumours such as telangiectasis venous angioma arterio venous aneurysm and angio-blastoma papilloma cholesteatoma psammoma, fibroma, chloroma and lipoma Primary sarcoma and actinomycosis are rare In children a tuberculoma is not so frequently met with as formerly Cysts include a dermoid hydatid cysticercus cellulosæ, serous cyst blood cyst, colloid cyst of the third ventricle and congenital cysts The glioma is ectodermal in origin it tends to infiltrate the cerebral substance, is vascular and liable to hæmorrhage or to cystic degeneration The meningioma arises from arachnoid connective tissue It invades bone with secondary bony overgrowth and com presses the brain Carcinoma of the brain is usually secondary to a growth in the breast lung or kidney The increase in size of the tumour causes cerebral anæmia, and the anoxia thus produced may result in localised epileptiform fits Increased intracranial tension and hydrocephalus are obstructive phenomena The frontal region is the site of about 20% of brain tumours and about 17% occur in the cerebellum

**Clinical Findings** The initial symptoms are usually insidious but at times an intracranial emergency provokes an acute onset Thus a child may be apparently perfectly well when suddenly *he falls uncon scious* owing to a hæmorrhage into a glioma The symptoms and signs are divided into two groups general and focal Often the general symptoms are first noted, but in some cases especially in cerebello

pontine tumours and in the aged general symptoms are inconspicuous *The general symptoms and signs* are an index of raised intracranial pressure and include headache vomiting and papillœdema These three are present together in about 60% of all cases of cerebral tumours and headache alone in about 80% of all cases The headache At first this is paroxysmal and often worse on stooping or at night Later it becomes persistent The site of the pain rarely serves to localise the tumour The skull may be tender on percussion over the tumour The headache is due to stimulation of the dural branches of the V nerve by stretching of the dura The vomiting This is typically projectile occurring apart from meals at night or early in the morning At times it is not projectile and there is also nausea Vomiting results from stimulation of the medullary centre Papillœdema This may exist in a marked degree without visual disturbance later sight is blurred and blindness ensues with atrophy of the optic nerves The papillœdema is due to the pressure of the cerebro spinal fluid in the sheath of the optic nerve on the central vein of the retina It is almost always present in cerebellar fourth ventricle and temporosphenoidal lobe tumours The pulse tends to be slow the temperature subnormal and respiration of the Cheyne Stokes type With tumours of the posterior fossa there is nearly always tachycardia In some cases if the skull is shaved dilated veins are apparent in the scalp over the tumour and in children the sutures may be widened A radiogram of the skull may show thinning of the bone (beaten silver appearance) calcification of a meningioma or displacement of the pineal body In about 60% of adults over the age of 25 years the pineal body is sufficiently calcified to be shown on X ray examination It is situated in the mid line above and behind the sella Ventriculography or encephalography may reveal an obstruction in or displacement of the ventricular system of the brain Arterial enceph alography (angiography) will show blood vessels displaced by a tumour The electro encephalogram may also aid in the localisation of cerebral tumours Slow delta waves arise in the cerebral tissue surrounding the tumour when the tumour causes progressive destruction of cortical tissue Discrete and non progressive cortical lesions or deep tumours not affecting the cerebral hemisphere or the cortex are not likely to be unmasked by electro encephalography The site of origin of the ab normal delta waves can be localised by a series of electro encephalo grams Radio isotopes which concentrate in the cerebral tumour are being used for the localisation of brain tumours In all cases of cerebral tumour it is dangerous to perform lumbar puncture if the intracranial pressure is raised as there is risk of death from incarceration of the cerebellum and medulla in the foramen magnum A tumour distant from the pituitary fossa may cause signs of hypopituitarism The increased pressure of the cerebro spinal fluid distends the floor of the third ventricle and causes pressure on the pituitary *Focal symptoms and signs* vary according to the site of the tumour and will be considered regionally

1 *Pre frontal Region* An alteration in mentality may be noted There may be drowsiness lack of orientation for time or a tendency to

make jokes (*Witzelsucht*) At times yawning, stiffness of the neck muscles, tremor of the homolateral hand diminution of the heterolateral abdominal reflex frequent nose rubbing and incontinence of urine or fæces at night may be found The " grasp reflex " ( forced grasping " or groping ) may be demonstrable An object placed on the palm of the hand on the side opposite to the cerebral lesion between the thumb and first finger, causes a grasping movement A similar ' grasp reflex " in the foot may sometimes be elicited with a tumour situated in the medial aspect of the frontal lobe and in the corpus callosum The ' grasp reflex " of the foot is elicited by light pressure applied to the plantar surface of the foot especially in the area of the ball of the big toe The reflex consists in flexion and adduction of the toes, of a tonic nature lasting for 15 seconds or longer There may also be inversion of the foot The reflex is normally present in all infants under the age of 9 months and disappears in the vast majority of cases by the age of 2 years It is present abnormally when a lesion interferes with the fronto pontine fibres It is therefore probably a release phenomenon and not a reflex and consists of low grade volitional movements The movements do not occur in infants during sleep nor in adult patients when unconscious Pressure symptoms include anosmia resulting from pressure on the olfactory nerve or bulb, and central scotoma with primary optic atrophy may occur on the side of the lesion and papillœdema in the other eye (Foster Kennedy syndrome)

2 *Ascending Frontal Region* Irritative or paralytic phenomena may be noted A cortical tumour may cause Jacksonian epilepsy with movements starting at the angle of the mouth or in the thumb or big toe on the opposite side There may be conjugate deviation of the eyes away from the affected side With subcortical tumours there may be paralysis of an arm leg or part of the face With left sided lesions aphasia may be present

3 *Parietal Region* The patient may notice an awkwardness in movement or blunting of sensation on the opposite side of the body Pain and temperature sensations are not affected but there may be loss of postural sense in the opposite limbs with astereognosis and disability in the discrimination of two points as judged by the compass test Word blindness may also result from a tumour involving the supra marginal or angular gyri on the left side

4 *Temporo sphenoidal Region* If the uncinate gyrus is involved there may be uncinate fits the patient passing into a dreamy state with unpleasant sensations of taste or smell and at times movements of the lips The growth may extend into the optic radiation with resultant quadrantic homonymous hemianopia When the first left temporal convolution is affected the patient may experience difficulty in naming objects, although he recognises the correct name when it is said (nominal aphasia)

5 *The Occipital Region* Involvement of the area around the calcarine fissure may result in homonymous hemianopia Visual hallucinations such as flashes of light may also occur

6 *The Internal Capsule* Involvement of the anterior limb causes

hemiplegia on the opposite side If the tumour affects the posterior limb there will be contralateral hemianæsthesia or hemianopia

7 *The Optic Thalamus* A tumour destroying the thalamus may result in the "thalamic syndrome" There is weakness of the opposite side of the body and choreic or athetoid movements may be seen Severe pains may be felt on the opposite side of the body and sensation is altered Such stimuli as tickling or scraping or the extremes of heat and cold may provoke very severe reactions although the patient is not able to differentiate between the blunt and sharp end of a pin There is also astereognosis and loss of postural sense on the opposite side

8 *The Third Ventricle* A colloid cyst produces intermittent symptoms especially very severe headache which may terminate in unconsciousness There is papillœdema with gradual failure of vision

9 *The Mid brain* A tumour in the region of the anterior corpora quadrigemina may cause bilateral ptosis with weakness of up and down movements of the eyes and a sluggish pupil reaction There may also be hemiplegia on the opposite side and bilateral ataxy of the arms if the decussating superior cerebellar peduncles are involved Bilateral deafness may result from involvement of the posterior corpora quadrigemina

10 *The Pons* The V VI or VII nuclei may be affected with paralysis of the muscles supplied by them There may also be hemiplegia and possibly hemianæsthesia on the opposite side of the body The pupils are often small due to interference with the impulses which cause dilatation These pass down the mid brain and cord to emerge in the cervical sympathetic

11 *The Medulla* There may be unilateral or bilateral paralysis of the IX X XI and XII nuclei with disturbance of swallowing mastication and speech Both pyramidal tracts may also be involved with bilateral hemiplegia

12 *The Interventricular Region* It is not usually possible to diagnose during life tumours arising here

13 *Cerebellar and Cerebello-pontine Regions* Tumours of these areas are considered on p 374

14 *The Pituitary Body* These tumours are considered on p 704

Differential Diagnosis The diagnosis of a cerebral tumour is often very difficult Thus localised or generalised convulsions may occur for years with a subcortical tumour These are usually considered to be due to epilepsy or to hysteria until papillœdema appears Vomiting may also be considered a hysterical manifestation Papillœdema may be met with in nephritis, septicæmia or severe anæmia When there are localising signs in the brain other conditions such as a vascular lesion or an abscess must be considered An abscess may run an afebrile course but the blood usually shows a leucocytosis In acute cases the acute variety of disseminated sclerosis encephalitis lethargica hypertensive encephalopathy lead encephalopathy hydrocephalus or abscess secondary to bronchiectasis may all require to be

eliminated The Wassermann reaction is helpful in establishing a diagnosis of a gumma, especially if the response to anti syphilitic *treatment is good* Secondary malignant deposits in the brain are suggested by the discovery of a primary focus elsewhere

**Course and Complications** The course is usually steadily progressive, but sudden exacerbations of symptoms due to œdema may be expected In some cases improvement occurs owing to absorption of œdematous fluid Complications include internal hydrocephalus hæmorrhage meningitis and secondary hypopituitarism

**Prognosis** This is always very grave and the outlook if surgery is adopted is usually gloomy Cerebral tumours as a class do not lend themselves to excision owing to their ill defined margins The most favourable as judged by the survival period after operation is the pituitary adenoma for over 70% of patients survive the operation by 7 years Next come the cerebellar astrocytoma the acusticus tumour and the meningioma The medulloblastoma is malignant Extra cerebral tumours are more amenable to removal without damage to the brain An operation which removes the tumour but results in the patient becoming hemiplegic and aphasic can neither be considered successful nor justifiable The outlook is hopeless with secondary malignant tumours A gumma responds very poorly to medical treatment and a tuberculoma may at times become obsolete

**Treatment** The Wassermann reaction should be determined and if positive a course of treatment given as described on p 596 If the tumour is localised accessible and of suitable type an attempt may be made to remove it surgically Decompression alone is a palliative measure to save sight and relieve headache In any case in which an operation is considered inadvisable the effect of a course of iodides should be tried Headache vomiting and semi coma may be temporarily relieved by the rectal injection of 8 fl oz of 25% mag sulph solution or by the intravenous injection of 25 ml of 30% sod chlorid solution or of 100 ml of 50% sucrose solution These intravenous injections should be given at the rate of 3 ml a minute Pain is relieved by aspirin gr 10 t d s or by pethidine mg 100 or morphine gr 1/4 injected as required Vascular tumours such as a venous angioma and arterio venous aneurysm should be treated by deep X rays

### Abscess of the Brain

**Definition** Localised suppuration in the brain

**Etiology** The abscess may be due to 1 Direct spread of infection This is the commonest cause The septic focus is most often in the ear as in chronic otitis media or in the mastoid cells In other cases the abscess is secondary to infection of the frontal ethmoidal or sphenoidal sinuses Syphilitic or tuberculous caries of the skull and erysipelas or a carbuncle on the scalp face or neck may lead to cerebral abscess 2 Blood borne infection The septic focus is frequently intra thoracic such as bronchiectasis lung abscess or empyema In other cases the cerebral abscess is secondary to osteomyelitis

puerperal sepsis infective endocarditis pyonephrosis etc 3 Local trauma The brain may be injured by a gun shot wound or a stab In some cases an abscess follows a blow on the head although neither skull nor scalp are severely damaged 4 Local lesions A cerebral neoplasm or tuberculoma rarely suppurates A cerebral abscess is most often met with between the ages of 11 and 35 years

Pathology A solitary abscess is usually due to trauma or to intra thoracic suppuration and multiple abscesses to extra thoracic suppuration A localised intracranial abscess is also found either extradurally between the dura and the skull or subdurally between the dura and pia The cerebral abscess may be acute or chronic The acute abscess contains creamy or blood stained material the chronic abscess is filled with greenish offensive pus and usually has a capsule formed by inflammatory tissue Various organisms such as staphylococci pneumo cocci streptococci and the Bact coli may be present or the contents may be sterile The Actinomyces bovis (Streptothrix actinomyces) is rarely found The abscess varies in size from a minute spot to that of a large orange It usually forms in the white matter below the cortex which is comparatively avascular between the brain tissue supplied by the end branches of the cortical and central cerebral arteries When the cerebral abscess is secondary to infection in the soft tissues outside the skull the infection may spread along thrombosed emissary veins An abscess of the brain secondary to osteitis of the skull is usually due to spread of infection along the perivascular spaces

Clinical Findings There is often a history that the patient has suffered from chronic suppurative otitis media for some years The onset of abscess formation may be heralded by cessation of discharge from the ear In some cases certain stages can be recognised clinically These are 1 *The stage of onset* This may be of short duration only 1 or 2 days during which the patient does not feel well there may be shivering or even a rigor with headache and perhaps nausea or vomiting The temperature rises to 101 F or higher but the pulse remains comparatively slow 2 *The latent stage* This may last for a few weeks The patient feels drowsy and complains of a dull headache and his appetite is poor

*On Examination* Local tenderness may be found on tapping the skull at some point The pupil may be dilated on the affected side Mental changes such as delusions hallucinations irritability etc have been described in some cases The temperature is usually low but if it is charted every 2 hours an irregular swing may be detected just above or below the normal line The pulse may drop to about 60 for short periods during the 24 hours The blood There is usually a leucocytosis of 15 000 or 20 000 per c mm The cerebro spinal fluid This is often under increased pressure and there is an increase of polymorphonuclear cells The chloride figure may be low The fluid is sterile The use of sulphonamides and of penicillin in the treatment of infections predisposing to cerebral abscess has very largely eliminated the preliminary stages The first symptom may be headache followed by a convulsion 3 *The stage of localised signs* Whether or not localising signs appear

depends upon the site of the abscess If it is in the tempero sphenoidal lobe there may be deafness on the opposite side or if it is on the left side of the brain there may be word deafness the patient not appreciating the meaning of spoken words, or having difficulty in naming objects Pressure on the pyramidal tract in the pons may result in weakness on the opposite side of the body with an extensor plantar response or loss of the abdominal reflex on the other side of the body Extension forwards may cause weakness of the lower part of the face on the opposite side In some cases there is pressure on the III or VI cranial nerves on the same side An abscess in the occipital lobe may cause homonymous hemianopia whereas if it is situated in the parietal lobe the patient may lose the power of recognising the shape of objects by touch (astereognosis) An abscess in the pre rolandic area will cause motor paralysis of the opposite side of the body The symptoms of a cerebellar abscess are described on p 374 Some degree of papilloedema may develop 4 *The terminal stage* This usually implies the phase of coma The abscess may rupture intraventricularly with convulsions delirium a high temperature and rapid action of the heart followed by death in a few hours Meningeal rupture is indicated by signs of meningitis

**Differential Diagnosis** It is often extremely difficult to diagnose with certainty the presence of a cerebral abscess in cases of chronic suppuration in the ear Mastoiditis and meningitis must be excluded and they may co exist with cerebral abscess The examination of the cerebro-spinal fluid establishes or excludes the presence of meningitis Further sinus thrombosis may cause difficulties in diagnosis There is usually a sudden onset to the symptoms in sinus thrombosis with rigors a high temperature rapid pulse, and at times local tenderness With a cerebral tumour papilloedema is likely to occur early and to be more intense than is the case with a brain abscess Leucocytosis is generally absent and no primary cause for an abscess is discovered A hæmorrhage into a glioma may cause acute symptoms resembling those produced by a brain abscess

**Course and Complications** Acute abscesses are usually rapidly fatal A chronic abscess may be encapsuled for years and give rise to no symptoms until it suddenly bursts into a ventricle or into the subarachnoid space Complications include pyocephalus hydro cephalus cerebral sinus thrombosis meningitis and septicæmia

**Prognosis** A cerebral abscess which causes symptoms will result in death unless drained surgically The mortality rate has been lowered to about 10% by the use of antibiotics

**Treatment** This is surgical In acute abscesses better results are obtained by aspiration than by drainage Crystalline sodium penicillin G 100 000 units per ml may be injected into the abscess cavity after aspirating some pus and its extent outlined by radiography after injecting into it 1 or 2 ml of a contrast medium such as diodone Cure can be effected in some cases by repeated aspirations with penicillin replacements In addition a course of intramuscular injections of 60 000 units of crystalline penicillin should be given every 3 hours for 7 to 10 days A chronic abscess may be drained immediately

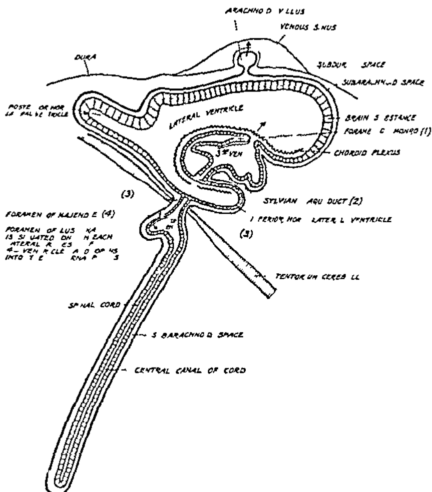



FIG 29 DIAGRAM INDICATING THE STRUCTURES CONCERNED IN THE PRODUCTION OF HYDROCEPHALUS

*Obstruction at* (1) foramen of Monro causes distention of one lateral ventricle

*Obstruction at* (2) Sylvian aqueduct causes distention of both lateral ventricles and of the 3rd ventricle

*Obstruction at* (3) tentorium cerebelli causes communicating hydrocephalus with distention of the lateral 3rd and 4th ventricles a communication being patent through the foramina of Majendie and Luschka with the subarachnoid space around the cord and the ventricular system The fluid passing from the 4th ventricle to the subarachnoid space cannot reach the subarachnoid space above the tentorium where absorption normally chiefly occurs

*Obstruction at* (4) foramina of Majendie and Luschka causes non communicating distention of the lateral 3rd and 4th ventricles

## Hydrocephalus

**Definition** Distension of the ventricles of the brain with cerebro spinal fluid

**Etiology** Theoretically the distension might arise from various causes such as 1 Over production of cerebro spinal fluid 2 Blockage of an exit from a ventricle 3 Obstruction to the absorption of cerebro spinal fluid The majority of cases in practice are due to 2 or 3 *Predisposing causes* 1 Age Usually infants or young children 2 Heredity Congenital hydrocephalus may run in families

In order to understand how these causes operate it is necessary to outline the physiology of the circulation of the cerebro-spinal fluid

**Physiology and Pathology** The cerebro-spinal fluid is derived probably by filtration from the blood circulating through the choroid plexuses in the lateral third and fourth ventricles These vessels lie invaginated in folds of the pia mater The fluid leaves the fourth ventricle by the foramina of Majendie and Luschka which are situated in the roof and lateral recesses of the ventricle and passes out into the subarachnoid space which here constitutes the cisterna magna It then circulates around the brain and cord In order to reach the brain it passes through a channel the chiasma tentorii in the subarachnoid space at the level of the tentorium cerebelli and so reaches the cisterna basalis and passing thence over the brain absorption occurs again probably by filtration through the arachnoid villi into the venous sinuses of the skull Hydrocephalus may be congenital or acquired and certain varieties are described

*External Hydrocephalus* This is a misnomer serous fluid accumulates in the subdural space leading to atrophy of the brain as in senile atrophy or general paralysis of the insane The cerebral ventricles may also be dilated constituting compensatory hydrocephalus

*Internal or Hypertensive Hydrocephalus* (see Fig 29) 1 *Increased production of fluid* It is possible as mentioned above that obstruction to the venous circulation in the brain would increase the output of fluid from the choroid plexuses to the ventricles Thus the great vein of Galen which drains the choroid plexuses of the lateral and third ventricles might be compressed by a subtentorial tumour 2 *Obstructed circulation of fluid* Obstruction of one foramen of Monro will cause dilatation of the corresponding lateral ventricle This may be due to a tumour of the choroid plexus and it may be intermittent Obstruction of the Sylvian aqueduct as by a tumour will result in dilatation of the third ventricle and both lateral ventricles Obstruction of the subarachnoid space as by meningeal adhesions at the level of the tentorium cerebelli causes a communicating hydrocephalus The fluid which leaves the fourth ventricle through the foramina of Majendie and Luschka cannot pass above the tentorium to be absorbed Normally only one fifth of the absorption takes place from the sub arachnoid space below the level of the tentorium and four fifths above this plane The pressure therefore rises both in the ventricles which dilate and in the subarachnoid space around the cord as shown by lumbar puncture Obstruction of the foramina of Majendie and

Luschka, as by meningeal adhesions, causes dilatation of the fourth, third and the lateral ventricles 3 *Defective absorption* This is not often a cause of hydrocephalus It may result from an inflammatory obstruction of the arachnoid villi by a generalised increased intravenous pressure in the skull due to a tumour, or by thrombosis of the superior longitudinal sinus (see otitic hydrocephalus p 813)

In congenital hydrocephalus a structural defect may be present, such as a Sylvian aqueduct with an opening in its roof, or a scar may cause obliteration of the aqueduct or the subarachnoid space may be obliterated by adhesions resulting from a hæmorrhage at birth Less often the foramina of Majendie and Luschka are obliterated by adhesions In many cases no cause is found In acquired hydrocephalus the lesion may result from syphilitic or meningococcal meningitis cerebral tumours especially those situated in the posterior cerebral fossa and rarely from thrombosis affecting the great vein of Galen or the cerebral venous sinuses The normal amount of cerebro spinal fluid present is about 150 ml (5 oz ), with hydrocephalus this is usually increased 3 or 4 times but much larger quantities have been found The lateral ventricles may be greatly dilated, the brain substance being reduced to a thin layer

**Clinical Findings** *Congenital Hydrocephalus* The head may be abnormally large at birth causing difficulty in labour, or the enlargement may not be noted until a few days after birth The skull bones become separated the scalp is thin and translucent, the veins being prominent and the hair scanty The infant s face is dwarfed by the protruding forehead and the lower lids may partly cover the pupils of the eyes, owing to downward pressure on the eyes by the orbital plates of the skull In a severe case the infant cannot lift its head off the pillow Other congenital defects may be present, such as hare-lip cleft palate spina bifida and imperforate anus, etc The infant may suffer from convulsions and some degree of spastic paraplegia If the infant survives mental deficiency or blindness due to optic atrophy may be found

*Acquired Hydrocephalus* The patient may be a young child or an adult of any age If the hydrocephalus develops after the skull bones have firmly united there is no enlargement of the head, if bony union is not complete some enlargement may occur The patient complains of very severe headache, vomiting disturbance of vision and unsteadiness or giddiness

*On Examination* Papillœdema may be present, and in some cases there is weakness of the arms or legs of a spastic type with exaggeration of the deep reflexes and an extensor plantar response

**Differential Diagnosis** The diagnosis of congenital hydrocephalus usually presents no difficulty slight cases should not be mistaken for rickets In cases of acquired hydrocephalus in which there is no enlargement of the skull, the condition closely resembles that of cerebral tumour, which may co-exist Special tests may be employed to demonstrate the presence and the variety of hydrocephalus These include

1 Ventriculography and encephalography Air is injected into the

lateral ventricle either through the anterior fontanelle if unclosed or by trephining the skull Radiograms will show the air in the ventricles and indicate whether there is free communication between them Encephalography is performed by lumbar puncture removing about 6 to 10 ml of fluid and then injecting with the syringe a smaller volume of air This is repeated until about 50 to 100 ml of air have been injected The air passes up the subarachnoid space and infiltrates not only the subarachnoid space over the cerebral cortex but also passes into the ventricles A block at any point may thus be demonstrated 2 Pressure tests If a manometer is inserted into the lateral ventricle and another into the spinal theca the pressure should be the same if no obstruction is present In a communicating hydrocephalus the cerebrospinal fluid is often under increased pressure corresponding with the increased intraventricular pressure In non communicating hydrocephalus the intraventricular pressure is raised the spinal pressure being normal 3 Dye tests If 1 ml of neutral phenolsulphonphthalein solution is injected into the lateral ventricle and a lumbar puncture is performed 10 minutes later in a normal patient the dye is then found in the spinal fluid If the dye is present a red colour develops in the spinal fluid on adding 25% NaOH solution In obstructive non communicating hydrocephalus the dye does not pass to the spinal fluid

**Course and Complications** Congenital hydrocephalus may pursue a rapid course or be arrested at any stage In some cases the children grow up and are remarkably intelligent Acquired hydrocephalus is usually progressive but the course must vary with the cause

**Prognosis** In congenital hydrocephalus death may occur in a few weeks or months in other cases arrest takes place In acquired hydrocephalus arrest may occur or the disease cause death in a few weeks months or years The prognosis depends upon the cause cases due to tumours are usually hopeless

**Treatment** Treatment is surgical Various operations used to relieve hydrocephalus include ventricular drainage ventriculo cysternostomy third ventriculostomy and in communicating hydrocephalus arachnoid ureterostomy Deep X ray radiation may be applied to the skull if there is a radio sensitive tumour causing obstruction If the condition is syphilitic as suggested by a positive Wassermann test a course of anti syphilitic treatment should be given see (p 596)

## Aphasia

**Definition** Disturbances of speech of writing and of the understanding of spoken and written words due to cerebral lesions

**Etiology** The lesion is on the left side of the brain in right handed people and is usually a hæmorrhage thrombosis embolus tumour or abscess

**Physiology and Pathology** Nothing is known very definitely regarding the physiology of speech The older physiologists believed in definite cortical centres such as a motor speech centre connected with the area of the brain controlling the speech muscles a motor writing centre connected with the hand a sensory visual centre and a

sensory auditory centre connected with the eye and ear respectively the whole area comprising the speech zone (see Fig 30) The modern tendency is to regard the subcortical zone with its association fibres as being intimately concerned with speech and writing faculties Thus Broca in 1861 taught that aphasia is due to a lesion at the posterior end of the left inferior frontal convolution Wernicke (1874) postulated in addition to the motor centre in Broca's area a visual sensory centre in the left supramarginal and angular gyri and their subcortical zones and an auditory sensory centre in the left superior and middle temporal convolutions and the subcortical area Marie (1906) considered that true aphasia is due to a lesion in Wernicke's sensory zone and that

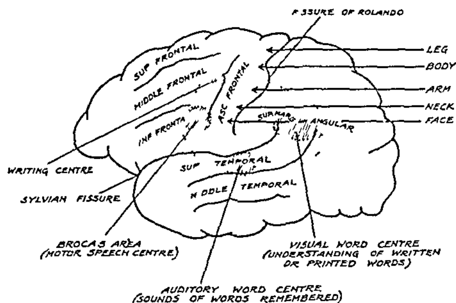



FIG 30 DIAGRAM OF THE SPEECH CENTRES AND CORTICAL MOTOR CENTRES OF THE BRAIN

motor aphasia results from a combination of anarthria (disordered articulation) with sensory aphasia Anarthria is due to a lesion in the neighbourhood of the lenticular nucleus and the genu of the internal capsule Thus in some cases of motor aphasia no lesion is found post mortem in Broca's area and in motor aphasia there is always some intellectual weakness Head (1910 and subsequently) after extensive study of the subject introduced certain terms for types of aphasia which are probably cortical in origin These include verbal aphasia with difficulty in word formation nominal aphasia with incorrect use of nouns in speech or writing syntactical aphasia with incoherent or jargon speech and semantic aphasia in which there is a lack of appreciation of the deeper significance of words and phrases More recent work by Goldstein (1948) is concerned with the complicated functional disturbance occuring in the various types of aphasia The speech area of the brain is supplied by the left Sylvian artery a branch of the middle

cerebral artery The usual lesion is a thrombosis embolus less often causes aphasia a hæmorrhage so frequently results in death that aphasia cannot be observed Aphasia of insidious onset may be due to a tumour Transitory aphasia may result from epilepsy migraine uræmia general paralysis of the insane or encephalitis lethargica

Clinical Findings In the majority of cases the aphasia has a sudden onset and is associated with other symptoms of a stroke There are various clinical types of aphasia such as 1 *Motor aphasia* The patient is unable to speak intelligibly but the actual muscles involved in speech are not necessarily paralysed Often he can say a few simple words and he can understand what is said to him 2 *Nominal aphasia* The patient may not be able to name an object which he recognises He can realise however whether or not it is correctly named by someone else 3 *Agraphia* The patient cannot write although the hand muscles which are controlled by the adjacent cortical area in the ascending frontal convolution are not paralysed 4 *Anarthria* Disordered speech may be due to a bilateral cortical lesion or a bilateral lesion of the internal capsule (pseudo-bulbar paralysis) or to a bulbar nuclear lesion 5 *Word deafness* Spoken language is not understood it seems to be a foreign tongue The patient s speech may be a jargon 6 *Word blindness* (alexia) The patient can see written words but does not appreciate their meaning 7 *Mixed aphasia* In the majority of cases the aphasia is mixed There is a disturbance of general intelligence as well as of speech writing and understanding of spoken words and often the patient is also suffering from hemiplegia

*Apraxia and agnosia* are conditions closely allied to aphasia In the former the patient is unable to carry out certain complicated movements such as buttoning up his clothes In the latter he confuses the use of objects

*On Examination* A certain routine should be observed in investigating cases of aphasia A complete neurological examination must be carried out and any lesion which is detected can often thus be localised An enquiry should be made as to whether the patient is right or left handed The power of speech should next be tested by asking the patient to say how he feels etc If he cannot speak intelligibly his power of understanding spoken words should be tested by asking him questions making him indicate his answers by signs or writing The power of understanding written or printed words is then determined by giving him a paper to read and asking him if he understands it He is asked to write to repeat words spoken to him to copy words pick out named objects to name objects and to write from dictation In this way the various connecting links of the speech area of the brain are tested

Prognosis This naturally depends upon the nature of the lesion If it is not a hæmorrhage or a tumour there is usually a tendency to improvement but subsequent attacks are liable to occur

Treatment When the patient has recovered from the shock and the immediate results of the lesion which has caused the aphasia his power of speech may be slowly improved by the help of a specially trained speech instructor

## Encephalitis Lethargica

*(Epidemic Encephalitis Type A)*

**Definition** A disease characterised by focal lesions in the grey matter of the brain and central nervous system often with a disturbance of the sleep mechanism

**Etiology** The causative agent is believed to be a filtrable virus the infection spreading from the naso pharynx to the arachnoid sac and so to the brain The virus has not been isolated Encephalitis lethargica occurred in epidemics but only sporadic cases are now met with The disease was noted in Vienna at the end of 1916 and described by von Economo Early in 1918 it appeared in London and was at first mistaken for botulism The virus may be wind borne direct trans mission from person to person is uncommon but it is thought to be slightly contagious Epidemics of encephalitis occurred in Japan from 1912 to 1929 and in the St Louis area U S A in the late summer of 1933 The Japanese encephalitis Type B is due to a filtrable virus which can be transmitted to monkeys The St Louis Type is due to a different virus which can be transmitted to monkeys and mice It is now endemic in America *Predisposing causes* 1 Age Usually between 15 and 45 years 2 Sex There is no distinction 3 Season Epidemics are liable to begin early in the winter 4 Locality Towns especially 5 Heredity The disease may be transmitted directly by a pregnant mother to the infant who develops it a few days after birth

**Pathology** Changes are found in the grey matter of the brain especially in the mesencephalon, basal ganglia and substantia nigra Thus the cranial nerve nuclei may be affected and less frequently the anterior horn cells in the cord Sleep regulation is believed to be dis turbed when the grey matter between the diencephalon and mesence phalon is affected At autopsy the brain often presents no naked eye changes but the leptomeninges may be hyperæmic and œdematous and on section the cortex and the basal ganglia may appear hyperæmic and actual hæmorrhages are sometimes seen in the grey matter Micro-scopically changes are found in the various masses of grey matter mentioned above There is a cuffing of the small vessels in the grey matter due to an infiltration of lymphocytes and plasma cells in the perivascular spaces Nerve ganglion cells may also undergo destruction in localised patches of grey matter The cerebral cortex is not usually affected

**Incubation Period** This is probably about 8 to 10 days

**Clinical Findings** Certain types of the disease are described The acute type, which was so common about 30 years ago is now seldom seen Sporadic cases start more insidiously

*Acute Cases* The three main types are 1 *The somnolent ophthalmo plegic type* There are fever drowsiness and oculomotor palsies The pyramidal tracts may be involved 2 *The hyperkinetic type* There are muscular twitchings and myoclonic or convulsive movements 3 *The amyostatic akinetic type* This is an acute form of Parkinsonism with

rigidity and absence of movement There may be tremors sialorrhœa and sleep inversion

Certain other forms of the acute disease will be enumerated such as 4 *The apoplectiform type* The patient is suddenly stricken as by a cerebral hæmorrhage and may die at once 5 *The cerebellar type* with marked ataxia 6 *The acute bulbar type* with disturbance of speech and swallowing 7 *The acute neuritic type* with paresis of the face and limbs 8 *The visceral type* with abdominal crises resembling those of tabes dorsalis 9 *The monosymptomatic type* Hiccough may last 5 to 7 days or there may be trismus or yawning 10 *The pseudo tabetic or pseudo paralytic type* with Argyll Robertson pupils and absent ankle and knee jerks 11 *The meningitic type* with symptoms of meningism 12 *The psychotic type* the initial acute delirium suggesting a psychosis

*Chronic Cases* The manifestations of the chronic form of the disease may directly follow an acute attack or they may only appear after an interval of several years In some instances the acute stage of the disease was so slight that it was not recognised as anything beyond a mild feverish illness or a temporary diplopia The symptoms of the chronic disease are due to destruction of nerve cells and take the form either of Parkinsonism or of juvenile pseudo pyschopathia *Parkinsonism* Both children and adults are affected The appearance of the patient resembles that described for paralysis agitans but the tremors and pill rolling movements of the hands are not often seen The skin is typically greasy and salivation may be very distressing The patient usually becomes progressively worse each winter but in a few cases the disease does not progress Tonic eye fits or oculogyric crises occur in some cases The eyes usually are directed upwards and sideways with convulsive movements Various tics or choreiform movements may also be a manifestation of the chronic disease and in some patients there is a tendency to obesity or polydipsia and polyuria

*Juvenile Pseudo pyschopathia* is another very distressing condition The child is demoralised although his intelligence is good He will commit every manner of evil such as lying stealing and acts of violence Although he often knows he is doing wrong he is powerless to control himself He is often particularly excited at night and has been described as the apache type of child

**Differential Diagnosis** As there is no specific test for the disease and as it exists in so many different forms a diagnosis can only be made by appreciating the nature and possible sites of the lesion and the various clinical pictures which may result Other conditions which require exclusion are polioencephalitis botulism drug poisoning uræmia meningitis cerebral tumour cerebellar lesions typhus or typhoid fevers post vaccinal or measles encephalitis influenza and paralysis agitans The cerebro spinal fluid should be examined in every case In botulism the pupils are usually dilated the mouth is dry and there are digestive disturbances Polioencephalitis (see p 424) In acute cases which are rapidly fatal it is usually impossible to make a diagnosis during life The young age incidence of the Parkinsonian syndrome is of great diagnostic value

**Course and Complications** The course depends upon the severity of the disease The acute stage may be mild and transitory but followed later perhaps at an interval of 4 to 9 years, by Parkinsonism A severe case may rapidly improve or relapses ensue during a prolonged illness or the case insidiously passes into a chronic stage The important sequelæ are the Parkinsonism and the pseudo psychopathia

**Prognosis** The mortality varies in different epidemics but the average figures show a death rate of 40% 30% become chronic cases and 30% recover completely The hyperkinetic type is the most fatal Death usually results from bulbar paralysis, cachexia inter current infection, or pneumonia

**Treatment** *Prophylactic* During an epidemic it is advisable to gargle night and morning with a 1 in 1,000 solution of potassium permanganate

*Curative* Acute cases should be isolated in bed No specific cure has been found If the patient is unconscious a lumbar puncture should be performed and the fluid allowed to escape slowly if it is under increased tension Sedatives will be required for the hyperkinetic type of disease, the best being an injection of hyoscin hydrobrom gr 1/100 once or twice a day This is also of value in relieving hiccough In milder cases aspirin may be given in doses of gr 10 every 4 hours The treatment of Parkinsonism is as described on p 356 For diurnal somnolence ephedrine hydrochloride gr ½ should be given at 8 a m , and for nocturnal insomnia Medinal gr 5 may be prescribed at night If sialorrhoea is a very troublesome feature the parotid region may be exposed to radiation by X rays For oculogyric crises Prominal (methylphenobarbitonum) gr 3 tablet may be used One or one and a half tablets daily may lessen the frequency of the attacks Benzedrine sulphate (amphetamina sulphas) mg 5 tablets by mouth 4 to 12 daily may relieve the fatigue depression and oculogyric crises It should be administered with caution and not given if there is arteriosclerosis Children suffering from pseudo psychopathia are best treated in special institutions

## *Acute Disseminated Encephalomyelitis*

**Definition** Inflammation affecting the brain and spinal cord

**Etiology** Certain types are described 1 Post vaccinal A complication of vaccination against small pox and rabies 2 After infectious fevers such as measles chicken pox mumps small pox scarlet fever diphtheria and whooping cough 3 Spontaneous with no known cause

**Pathology** The surface of the brain may appear congested and on section punctate hæmorrhages may be seen Microscopically there is a diffuse infiltration of the perivascular spaces with round cells and plasma cells and demyelination of the white matter may be present around the vessels The pons medulla and lumbar part of the cord are especially liable to be affected

**Clinical Findings** 1 *Post vaccinal Encephalomyelitis* This was first described in 1907 and again in 1912 (see also p 575) The symptoms usually begin 10 to 12 days after a primary vaccination in a child over

the age of 1 year or in an adult The risk of post vaccinal encephalo myelitis under the age of 1 year is negligible The patient complains of malaise headache and vomiting The temperature rises to over 102° F the legs become paralysed and the plantar responses may be extensor There may be neck rigidity convulsions and trismus The cerebro spinal fluid pressure is increased and there may be an excess of protein and of mononuclear cells There is later incontinence of urine and fæces in cases which die in coma The patient may recover completely or die in a day or so or recovery may be incomplete with residual states such as hemiplegia

2 *Encephalomyelitis associated with Infectious Fevers* The symptoms occur as a complication of measles mumps small pox and chicken pox the legs may rapidly become paralysed and there is retention of urine The paralysis may be flaccid with diminished reflexes and later spastic with increased reflexes and an extensor response There may also be dissociated anæsthesia of the legs pain and temperature sense being lost and touch vibration and postural sense being unaffected In other cases there are convulsions with headache The cerebro spinal fluid This is often normal but in some cases there is an excess of cells and protein

3 *Spontaneous Encephalomyelitis* The patient is usually a young adult who is suddenly taken ill either with pains in the body or limbs or with weakness of the legs There is often a spastic paresis or para plegia with exaggerated or lost deep reflexes and usually an extensor plantar response There may be sensory loss in one or both legs or a Brown Séquard syndrome with paralysis of one leg and loss of sensation on the other side There is usually retention of urine The cranial nerves are seldom affected but there is often nystagmus In some cases meningeal symptoms are present or there may be aphasia and hemi plegia The cerebro spinal fluid is normal

**Differential Diagnosis** The disease is probably distinct from acute disseminated sclerosis Thus the fever shooting pains loss of pain and temperature sensations the normal cerebro spinal fluid and at times loss of deep reflexes in the legs during the acute stage and euphoria are *in favour of encephalomyelitis* *In acute disseminated sclerosis there* is more likely to be diplopia loss of postural and vibration sensations in the leg and retrobulbar neuritis Other conditions which may require exclusion are polioencephalitis encephalitis lethargica and meningitis

**Course and Complications** The disease may pursue a rapidly fatal course or there may be recovery after an acute illness lasting 1 or 2 weeks Sequelæ include a persistent Brown Séquard syndrome a condition resembling chronic disseminated sclerosis hemiplegia or aphasia

**Prognosis** The mortality rate varies from 10% to 50%

**Treatment** The patient must be kept in bed and symptomatic treatment applied In post vaccinal encephalomyelitis 5 to 80 ml of serum obtained from an individual successfully vaccinated 14 days previously should be injected intravenously or into the patient s theca

after lumbar puncture If the patient is comatose 25 to 100 ml of 25% sucrose solution should be injected intravenously In encephalomyelitis following measles 10 ml of convalescent serum should be injected intramuscularly and repeated if necessary The value of serum treatment is very doubtful

## Encephalitis Periaxialis Diffusa

### (*Schilder's Disease*)

**Definition** A disease characterised by inflammatory changes in the white matter of the brain with demyelination of nerve fibres

**Etiology** This is a rare disease the cause of which is unknown Children and young adults are chiefly affected

**Pathology** The brain often appears small On section, sharply defined patches of greyish or reddish brown hyaline appearance are seen in the white matter The occipital and temporal lobes are especially likely to be affected Lesions also occur in other parts of the cerebrum and these may coalesce The cerebellum is sometimes affected Microscopically it is seen that the myelin of the white matter is destroyed with secondary degeneration of the axis cylinders An infiltration of round cells and neuroglial cells is present in the white matter, and *phagocytes and lymphocytes are gathered around the vessels*

**Clinical Findings** The patient is often a child about the age of 8 or 10 years The symptoms depend upon the area of brain affected in a typical case the patient is comparatively suddenly taken ill with malaise and headache There may be early disturbance of vision giddiness, deafness or difficulty in walking or in using an arm

*On Examination* If the cortical motor area is involved there may be spastic weakness of a leg or arm with exaggerated deep reflexes and an extensor plantar response With involvement of the occipital cortex there is dimness or loss of vision Thus both visual fields may be totally *blind or with unilateral lesions there is a homonymous hemianopia* The pupils react normally and the discs are normal occasionally there is a slight degree of optic neuritis When the frontal lobes are affected there may be alteration in mentality such as childishness or actual dementia In other cases there is disturbance of speech or aphasia Jacksonian fits or generalised epileptiform convulsions may occur The cerebro spinal fluid This is normal The temperature is usually normal but may rise before death

**Differential Diagnosis** The diffusely scattered and spreading nature of the lesions usually serves to differentiate the disease from a *cerebral tumour* *With the latter* papilloedema is generally more intense

**Course and Complications** The course is usually steadily progressive arrest being very rare

**Prognosis** The disease is usually fatal either in a few months or in 2 to 3 years

**Treatment** No cure is known

### Cerebral Diplegias

*(including Little s Disease)*

**Definition** A disease of infants characterised by spastic diplegia or spastic rigidity of the arms trunk and legs

**Etiology** In Little s disease the causes are operative before or during birth They include birth injuries resulting in meningeal hæmorrhage asphyxia at birth hæmorrhagic disease of the new born intra uterine infections and faulty development (agenesis) of the pyramidal tracts In other cases of cerebral diplegia the spasticity develops in early life often due to encephalitis and rarely to hypo thyroidism Infantile hemiplegia may be due to hæmorrhage infections and possibly to degenerations

**Pathology** Cerebral venous hæmorrhage or petechial and confluent hæmorrhages may be found post mortem In other cases the con volutions of the brain appear atrophied on naked eye examination (atrophic lobar sclerosis) and microscopically atrophy or sclerosis of the pyramidal tracts is seen *Multiple small cysts* (porencephaly) are at times present in the brain

**Clinical Findings** No symptoms are usually noticed at birth The infant does not begin to walk until after the normal age (1 year) It is then noticed that the legs are weak

*On Examination* There is spasticity of the legs with an increase of the deep reflexes The extensor plantar response of the infant is perpetuated Later the heels become raised and the child cannot walk on them the legs are adducted and may actually cross one another on walking (scissors gait) The arms are rarely affected In the majority of cases the mentality of the child is normal In other cases mental deficiency, athetoid movements or epileptiform fits may occur There is usually a tendency to improvement but death may result from some intercurrent disease

**Treatment** In the early stages massage and passive movements should be applied to the affected limbs Later contractures may be improved by operations such as tenotomy Active remedial exercises are useful in the co ordination of movements The administration of thyroideum in doses of gr 1/10 t d s is of value in many cases

### Amaurotic Family Idiocy

*(Cerebro macular Degeneration Tay Sachs Disease)*

**Etiology** The cause is unknown The patient is nearly always a Jewish infant It tends to affect several members of one family

**Pathology** There is degeneration of nerve cells with deposition of lipoid material throughout the brain and cord The nerve cells swell and the nuclei are destroyed

**Clinical Findings** The infant appears healthy at birth but between the age of 3 and 6 months muscular weakness is noticed This often starts in the back and becomes generalised Ophthalmoscopic examination shows a condition of primary optic atrophy with a cherry

red spot at the macula due to atrophy of the retina and exposure of the choroid The disease is progressive there is marked mental deterioration and death occurs in about 6 to 12 months from the onset of symptoms

*The Juvenile Type* (*Spielmeyer Vogt s disease*) Another type of the disease affects young children usually not Jews There is some degree of optic atrophy with retinitis pigmentosa but the red spot does not appear at the macula

## Tuberous Sclerosis

### (*Epiloia*)

**Definition** A congenital disease characterised by mental deficiency, epileptic attacks and adenoma sebaceum

**Pathology** Nodules resembling glial sclerosis are found in the cerebral cortex and in the walls of the lateral ventricles These may calcify Various tumours are also met with in the retina kidneys heart or lungs

**Clinical Findings** Convulsions are usually noticed during the first year of life The adenoma sebaceum appears a few years later first in the labio nasal folds it spreads to the cheeks in a butterfly area involving later the chin but not the upper lip The adenomas tend to coalesce and become dark red or brown Café au lait staining may be seen elsewhere on the skin and a shagreen patch in the lumbo sacral region There may also be neurofibromatosis X ray examination of the head shows thickening of the skull and calcified nodules in the brain or projecting into the lateral ventricles The long bones hands or feet may show periosteal thickening osteoporosis or cyst formation

**Prognosis** Death may be delayed for many years, and then result from intercurrent infection epilepsy or renal tumour

**Treatment** This is devoted to care of the patient on account of the mental deficiency and to treatment for the epileptic fits

## Epilepsy

### (*Idiopathic Epilepsy*)

**Definition** A chronic disorder in which there are recurrent attacks of unconsciousness with or without convulsions or in which there are psychic seizures due, in the majority of instances to no *known cause*

**Etiology** In some cases an attack may be provoked by a fright *Predisposing causes* 1 Age The majority of cases begin in childhood or about the time of puberty In about 25% of cases epilepsy begins after the age of 20 2 Sex The incidence is equal 3 Hereditary transmission The disease is not transmitted but there is a hereditary trait which is transmitted in a definite proportion of cases 4 Dyspituitarism especially of the Frohlich type 5 Migraine may be a precursor 6 Alkalosis 7 Water intoxication 8 Menstruation 9 Anoxæmia and hypoglycæmia 10 Local cerebral lesions are present in a certain proportion of cases especially in temporal lobe epilepsy

**Pathology** The electro encephalogram shows that epilepsy is associated in many cases with the development of abnormal rhythms in the cerebral cortex It is a paroxysmal cerebral dysrhythmia in which discharges occur from living neurones This probably depends upon the metabolism of the neurones Post mortem no changes are found in the central nervous system of patients who have suffered from idiopathic epilepsy

**Clinical Findings** The patient is usually a child or young adult There may be a history of infantile convulsions The general health is often good but the patient is apt to be lazy egocentric and prone to lying The speech may be of the plateau type the vowels being pronounced with a peculiar even tone The clinical findings of idiopathic and organic epilepsy are indistinguishable and the so called Jacksonian or local fits are met with at times in many cases of idiopathic epilepsy in which there is no local lesion of the brain Certain types of epilepsy are described —

1 *Petit mal* (minor attacks) The patient may be quite unaware of their occurrence The onlooker notices that the patient suddenly stops speaking or remains motionless for a second or so He looks dazed the pupils may dilate the head moves slightly the eyes deviate and there may be yawning The face usually becomes pale and subsequently flushes The patient then comes round suddenly and continues what he was doing During the attack there is a brief loss of consciousness

*Pyknolepsy* This is a disease of children closely resembling petit mal The onset is usually between the ages of 4 and 12 years The attacks begin quite suddenly and are of very brief duration lasting only a few seconds The child has a vacant look and is unable to speak or move There may be twitching of the eyelids and the eyes may roll upwards There are no convulsions and the child does not fall and is not really unconscious Recovery is sudden and complete As many as 100 such fits may occur in a day The attacks usually disappear after puberty and are totally uninfluenced by drug treatment

*The akinetic form* The patient may suddenly fall to the ground without warning due to loss of postural control (*morbus caducens*) He gets up at once after a momentary loss of consciousness

*Myoclonic jerks* Sudden jerky movements occur of the arms or trunk without apparent loss of consciousness These three forms constitute the *petit mal triad*

2 *Grand mal* (major attacks) In about half the cases there is a preliminary phase known as an aura *The aura* This may take the form of auditory visual olfactory or gustatory sensations In other cases numbness or tingling may be felt in one of the extremities or a sensation of something travelling up the arm or leg There may be slight muscular twitchings cramp sweating giddiness or peculiar dreamy states or reminiscent sensations of familiarity with certain places with which the patient is personally unacquainted These auræ indicate the beginning of a fit in some portion of the brain and the process whether it be one of excitation or inhibition spreads rapidly

producing unconsciousness and convulsions These usually affect first such muscles as those which move the head and eyes and then rapidly become generalised The patient may give an initial unconscious cry and then fall to the ground There is marked pallor *The tonic phase* This lasts for about half a minute The muscles are contracted and the attitude is often that of decerebrate rigidity, the arms being flexed and adducted, the legs extended the head retracted and often turned to one side and the back arched Respiration usually ceases with resultant cyanosis The tongue is protruded and bitten as a result of masticatory spasm *The clonic phase* This is characterised by jactitations violent muscular contractions and relaxations producing convulsions They may be first noted on one side of the body Breathing is stertorous and frothy fluid exudes from the mouth which is blood stained if the cheeks or tongue have been bitten The face is contorted the mouth opening and closing and the eyes rolling upwards There is frequently enuresis and the pulse is rapid and of small volume The pupils are dilated and do not respond to light the corneal and deep reflexes are usually abolished and the plantar responses are often extensor It is believed that the blood pressure falls during an attack After 3 or 4 minutes the convulsions cease and the patient falls asleep or rapidly regains consciousness He does not remember the attack but may be made aware of it by finding that he has injured himself or that he is in some unusual position or that he has had incontinence of urine On recovering consciousness he may complain of severe headache or of nausea or vomiting There is frequently polyuria with a trace of albumin in the urine and an extensor response may persist for a few hours with increase of the deep reflexes In some cases there is a temporary aphasia or post epileptic paralysis (Todd s paralysis)

3 *Psychic seizures* (psychic equivalents psychomotor epilepsy) These include peculiar dream states with hallucinations of sight smell taste or sound There may be amnesic states, automatic actions chewing movements or stupor In the third variety there may be jerky movements rigidity actions of violence fugues or running episodes In temporal epilepsy there are psychomotor seizures with an aura which may be olfactory or abdominal There are often personality changes The electro encephalogram shows changes in the majority of patients suffering from epilepsy even between the attacks The abnormality between the fits consists in the occurrence of intermittent irregular slow waves Seizure waves occur during a fit in grand mal there are sharp spikes in petit mal quick sharp spikes alternate with slow round waves and in psychomotor attacks square flat waves are seen (see Fig 23) About 20% of epileptics have a normal electro encephalogram and in about 12% of normal people the electro encephalogram is abnormal although there are no fits

4 *Status epilepticus* The patient may pass into a state of coma interrupted by convulsive attacks without recovery of consciousness The temperature is raised to 104° or 105° F the pulse is frequent and of small volume there is rapid bodily wasting and death is likely to

occur in a few days from fatty myocardial degeneration or pulmonary œdema

**Differential Diagnosis** Idiopathic epilepsy must be diagnosed from 1 Infantile convulsions Clinically the convulsive phenomena are alike and epilepsy can only be excluded by the course of the disease infantile convulsions being of a temporary nature epilepsy tending to recur 2 Jacksonian epilepsy This type is in some cases organic and associated with a definite cerebral lesion such as a depressed fracture of the skull tumour of the brain gunshot wound of the head etc Local fits ensue In other cases local fits occur in idiopathic epilepsy 3 Epileptiform convulsions associated with uræmia congenital cystic disease of the kidney alcoholism lead poisoning cerebral syphilis general paralysis of the insane cerebral cysticercosis etc 4 Hysterical attacks During these attacks the patient is not completely unconscious The attacks occur in the presence of onlookers and the patient does not fall in a dangerous situation The corneal pupil and deep reflexes are usually present The patient does not bite his tongue nor micturate Hysterical attacks may alternate with epileptic ones 5 Syncopal attacks These are associated with a fall in blood pressure and are usually provoked by some unpleasant sight or thought There is loss of consciousness and convulsions may occur Adams Stokes attacks are associated with cerebral anæmia 6 Vasovagal attacks (medullary syndromes) These resemble a much prolonged attack of petit mal There is cutaneous pallor and sweating a slow and feeble pulse dilated pupils flaccid muscles abdominal cardiac or respiratory discomfort and the patient may fall 7 Narcolepsy This may be differentiated from epilepsy Males are affected slightly more frequently than females usually about the age of puberty or more rarely about the age of 40 and the disease is very obstinate It is characterised either by sudden attacks of uncontrollable sleep or by the cataplectic attacks with loss of muscle tone The latter are induced by emotions especially by amusement The patient falls but retains consciousness He is unable to speak or to move the lower jaw drops and the eyelids tend to close After a few seconds or minutes he suddenly recovers There may be protrusions of the tongue or contraction of the facial muscles possibly due to the patient trying to speak The patient usually describes the attack by saying that he flops In some cases narcolepsy is complicated by epilepsy Traumatic cases also occur following blows on the head and possibly result from minute hæmorrhages near to or in the third ventricle 8 The Kleine Levin syndrome There are periodical attacks of somnolence lasting 2 or 3 weeks accompanied by excessive hunger

**Course and Complications** The course of epilepsy is very variable Minor attacks may occur frequently and be interspersed with major fits Major attacks may recur at comparatively long intervals Complications include post epileptic phenomena such as automatic actions Thus the patient may undress after an attack being quite unaware of his actions Mania and paralysis may ensue Rupture of the heart and fracture or dislocation of the jaw may occur during the convulsive stage

Mental deterioration is especially likely to follow attacks which beg early in childhood, it does not depend upon the violence of the attacl

**Prognosis** The outlook is usually *unfavourable in epilepsy* as spontaneous recovery is comparatively infrequent The most favour able features are if the attacks begin after the age of 20 and if they a not very severe Mental deterioration is an unfavourable featu Petit mal is less influenced by treatment Status epilepticus is a ve grave event death may be due to an accident such as suffocatio during a nocturnal attack

**Treatment** *Prophylactic* An attack *may sometimes be aborte* when there is an aura of something travelling up the limb by tying bandage around the limb close to the trunk Firm muscular contractio by clenching the fist may at times avert an attack heralded by a recog nised aura In view of the transmission of a hereditary trait it inadvisable for epileptics to marry

*Curative* 1 During the attack No special treatment is usuall required for attacks of petit mal The immediate treatment for a attack of grand mal consists in loosening the patient s collar removin false teeth if present and preventing him from biting his tongue b inserting a spoon between his jaws The patient should not be arouse if he falls asleep after the attack If there is much collapse a hypodermi injection of a stimulant such as strychnin hydrochlor gr 1/60 may b given A patient who is subject to attacks of epilepsy occurring b night should sleep on a low bed

2 Between the attacks Bromides and belladonna are now seldon used for the treatment of epilepsy but they are occasionally effectiv when other methods fail

*Petit mal* Trimethyloxazolidinedione (Tridione) is of value Th dose for an adult is 0 3 G capsule t i d and this may be cautiously increased to 0 9 G t i d For a child 0 15 G is given t i d increased to 0 6 G t i d Side effects include photophobia a morbilliform rash aplastic anaemia and nephrosis This drug should be discontinued if a rash appears and a blood count should be done every month If Tridione fails dimethylethyloxazolidinedione (Paradione) may be tried in the same doses the same precautions being taken

*Grand mal* Treatment is usually begun with phenobarbitone first gr ½ once or twice a day increasing up to gr 1 t i d Drowsiness may be checked by the simultaneous administration of amphetamine sulph mg 5 before breakfast and before lunch *It is often found helpful* to reduce the dose of phenobarbitone and combine it with phenytoinum sodium (Dilantin Epanutin Eptoin) in doses of 0 1 to 0 2 G t i d for an adult and 0 01 to 0 1 G t i d for a child It is put up in capsules containing Epanutin 0 1 G (gr 1½) with phenobarbitone 0 05 G (gr ¾) Side effects include hypertrophy of the gums ataxia nystagmus diplopia skin rashes neutropenia and delusions

Primidone 5 phenyl 5 ethylhexahydropyrimidine 4 6 dione (Mysoline) is of value in grand mal and temporal lobe epilepsy but of less value in petit mal It may produce drowsiness and giddiness The dose for an adult is 0 25 G daily increased to 3 or 6 doses in the

24 hours In very resistant cases 5 ethyl 3 methyl S phenylhydantoin (Mesontoin) may be tried in doses for an adult of 0 2 G t i d It may produce drowsiness or aplastic anaemia and the blood should be examined monthly

Acetazolamide (Diamox) is an inhibitor of carbonic anhydrase and so causes a metabolic acidosis It is under trial in the treatment of epilepsy the dose being 8 to 30 mg /kg body weight usually given in one dose in the morning The total daily dose given varies between 250 and 1 500 mg

In temporal lobe epilepsy sclerotic areas may be present in the inferior and medial part of the temporal lobe Treatment has been recommended by excising the anterior part of one temporal lobe It is claimed that the fits are abolished or reduced in over 50% of cases so operated on It is too soon to decide yet whether this operation is of permanent value

For status epilepticus the patient should be kept in bed in a dark room and an immediate subcutaneous injection given of morphin sulph gr 1/6 or hyoscin hydrobrom gr 1/200 A subcutaneous injection of phenobarbitone sodium gr 2 in m 11 of water should then be given and if this does not stop the convulsions an enema may be administered followed by a rectal injection of 3/4 to 1 fl oz of paraldehyde (for an adult) with an equal quantity of olive oil and the temperature may be lowered by tepid sponging Alternatively 10 ml of paraldehyde may be injected intramuscularly for an adult This is put up in 10 ml ampoules

*General Treatment* The patient should be encouraged to take an interest in outside affairs as far as possible but mental worry and over fatigue are injurious A regular mode of living with open air exercise and avoidance of alcohol should be enforced No epileptic should be allowed to drive a car or engage in any occupation in which he may be a danger to himself or to others Children of the poorer class are often best treated in special institutions or colonies

*Narcolepsy* This may be treated on the same lines as epilepsy in some cases good results have been obtained by the administration of ephedrine hydrochloride gr 3/4 three times a day for an adult and gr 3/8 for a child or by amphetamine sulphate mg 5 tablets 2 to 18 daily

## Chorea

*(Sydenham s Chorea Rheumatic Chorea St Vitus Dance)*

**Definition** A disease characterised by spontaneous and irregular muscular contractions usually associated with an acute rheumatic infection

**Etiology** The cause is not definitely known but in the majority of cases chorea is believed to be due to a rheumatic infection It may be associated with scarlet fever diphtheria measles and rarely with chicken pox *Exciting causes* 1 Fright 2 Pregnancy especially during the first three months of the first pregnancy *Predisposing causes* 1 Age Childhood and adolescence rare after 25 2 Sex

Females predominate 3 Overwork at school and left handedness 4 Poverty and unhygienic surroundings 5 A familial rheumatic tendency

**Pathology** Changes are found in the cerebral cortex, the caudate nucleus the substantia nigra, the red nucleus and possibly the superior cerebellar peduncles are affected A focal lesion of the corpus Luysii (subthalamic body) will cause hemichorea on the opposite side Microscopically there is vasodilatation at times perivascular cuffing, and degeneration of ganglion cells

**Clinical Findings** The patient is often a child of about the age of 10 The child becomes nervous, irritable and unnaturally emotional She is listless, inattentive and has difficulty with her lessons There may have been loss of appetite, and sore throat growing pains or rashes on the skin (such as erythema nodosum) In some cases the child seems lame or drags one leg or is clumsy, and is apt to drop things and she may have difficulty in speech when excited Twitching of the face grimacing twitching of the hands or shrugging the shoulders may be the first sign noticed in other instances

*On Examination* The signs present vary with the severity of the case

*A Mild Case* The twitching movements are seen chiefly on both sides of the face and in one hand, arm or shoulder Tremors are seen when the arm is extended and there is a tendency for the arm on the affected side to droop with flexion of the wrist and over extension of the metacarpo-phalangeal joints The tongue may be jerked in and out when the patient is asked to protrude it Jerky movements may occur at the elbow and shoulder when the patient grips the examiner s hand The temperature is normal but the heart may be slightly dilated

*A Moderate Case* The movements now are very obvious and the child is hardly ever still The legs are less affected than the arms When only two limbs are involved they are always homolateral The face and trunk muscles are bilaterally affected The movements are described as spontaneous, large and irregular To a certain extent they can be controlled and they cease during sleep The affected muscles are lacking in tone and the arm does not swing on the affected side on walking Hypermetria is shown by the difficulty in grasping objects and by the finger overshooting the mark in the finger nose test Dysdiadokokinesis may also be present The reflexes The cutaneous reflexes are normal The deep reflexes are diminished in moderately severe cases and the knee jerks are sustained ' the leg being hung up for a brief period before it falls again The sphincters are not affected Sensation is normal The temperature is often normal unless the illness is complicated by an active carditis and the pulse is not so rapid as in rheumatic fever

*Severe Cases* (Chorea Gravis) The movements are violent and the patient may be thrown from a chair or bed There is disturbance of speech and of deglutition Maniacal symptoms may appear or there may be delirium with visual hallucinations The temperature is usually raised and signs of endocarditis are rarely lacking Other types

described include *Chorea mollis* in which there is a flaccid paresis of the voluntary muscles with only slight movements *Paralytic chorea* one arm or leg may be limp and almost useless

The electro encephalogram in chorea usually shows abnormalities In hemichorea there are waves indicating electrical activity in the opposite cerebral cortex

**Differential Diagnosis** The diagnosis of a typical case of chorea usually presents few difficulties Certain other conditions may require consideration such as 1 A tic Here the movements are repetitive and limited to certain muscles 2 Hysteria St Vitus dance as originally described, was presumably a hysterical manifestation The movements in hysteria have not the character nor distribution of those in chorea 3 Symptomatic chorea This occurs in such diseases as epilepsy infantile cerebral paralysis tabes dorsalis general paralysis of the insane or as a post encephalitic symptom 4 Encephalitis lethargica In the hyperkinetic variety of this disease there is generally a III nerve paralysis 5 Athetosis The movements here differ from those of chorea and are usually confined to the hand or arm They are generally the sequela of a hemiplegia 6 Huntington s chorea The age incidence is later there is a familial history and usually mental degeneration

**Course and Complications** Mild cases respond to treatment in a few weeks but if the condition is overlooked or the child is pressed at her work the disease is prolonged Recurrent attacks occur in about 30% of cases often after an interval of a year A watch should be kept for complications such as endocarditis or pericarditis Aortic disease rarely occurs Mitral stenosis often develops many years after an attack of chorea

**Prognosis** This is good except in chorea gravis and maniacal chorea In chorea gravis dysphagia is a dangerous symptom and maniacal chorea is usually rapidly fatal The gravity of the disease is increased by endocarditis or pericarditis Abortion is liable to occur in the chorea of pregnancy and may be followed by the death of the mother

**Treatment** In all cases the child should be taken away from school and kept quietly at rest at home preferably in bed unless the disease is very mild In severe cases precautions should be taken to prevent the child injuring herself by falling out of bed Adequate nourishment is necessary and milk is of value in this respect The bowels should be kept open daily Warm baths tend to relieve the nervous symptoms There is no specific curative drug Good results are usually obtained with calcium aspirin in doses of gr 5 to 15 or more t d s for a child of 10 to 12 years For restlessness phenobarbitone gr ½ to 1 may be given t i d and in more severe cases phenobarbitone sodium gr 2 in m 11 of water should be injected subcutaneously In maniacal chorea a mixture containing Pot brom gr 10 chloral hydrat gr 15 syr aurant m 30 aquam ad fl oz ½ should be given t d s for a child of 12 In some cases an injection of hyoscin hydrobrom gr 1/300 is of value If the convulsions are not checked an inhalation of chloroform may be necessary from time to time The results of the administration of

ACTH or of cortisone are very variable During the illness a watch should be kept on the heart to detect signs of dilatation, carditis or pericarditis Should they occur further rest in required Convalescence should never be hurried and the child *not be allowed to return to school until all movements have* No examinations should be allowed for a year after returning to

## Huntington's Chorea

### (*Chronic Progressive Chorea*)

**Definition** A disease characterised by choreiform movemen mental deterioration

**Etiology** The cause is unknown *Predisposing causes* 1 Her The disease presents a definite familial incidence and in some cas mental and physical characteristics are transmitted independ The original patient went from Bures in Suffolk to America in 163 one line was traced through 30 generations up to 1932 1 000 being noted It was described by Huntington of Pomeroy Ohio in In successive generations the disease tends to show itself earlier fu if one generation escapes the disease does not reappear 2 Usually between 30 and 50 years 3 Sex The incidence is equal

**Pathology** The brain is usually below the average size, with atrophy of the frontal lobes and corpus striatum the putam especially affected with secondary involvement of the globus pal The mental changes may be due to atrophy of the cortical cells

***Clinical Findings*** *Choreic movements are first noticed in the* or in the muscles moving the head The arms and legs are subsequ affected The speech may have a peculiar explosive character oculo motor muscles are usually the last to be involved The pa finally becomes chair ridden or bed ridden The mental change insidious, the patient is irritable and lacking in external interests gradually his higher mental faculties fail

**Differential Diagnosis** The character of the motor and me changes with a familial history usually establishes the diagnosis blood and cerebro spinal fluid should be examined to exclude ne syphilis Senile chorea The age of onset is later There are ch movements but no mental changes This may be an atypical for Huntington s chorea Congenital chorea This is present from b and is probably due to agenesis of the cells in the corpus stria *There is no spasticity of the limbs but the mental processes are* ra slow The condition appears to be related to Huntington s ch Apoplectic chorea This results from a hæmorrhage into the substa nigra in the mid brain The patient has choreiform movements usually dies in a week or so

**Course and *Complications*** *The course is usually slow* and patient may live for 20 years or so after the first appearance of sympto Homicidal or suicidal mania may occur as a complication

**Prognosis** The disease although not curable is not necessarily fatal and death is often due to an intercurrent infection

**Treatment** *Prophylactic* Marriage should be avoided by a sufferer from the disease

*Curative* No drug is known to have any effect in checking its course

## Pink Disease

*(Infantile Acrodynia Erythrœdema Tropho dermatoneurosis)*

**Definition** A disease of infants characterised by mental disturbances insomnia sweating disordered sensation of the extremities and peripheral vascular phenomena

**Etiology** The cause of the disease is unknown It has been attributed at times to a virus infection a deficiency of vitamin $B_1$ hypofunction of the adrenal cortex and more recently to mercury poisoning from tablets ointments or teething powders as mercury has been found in the urine in Pink disease Functional overactivity of the adrenal medulla has also been postulated *Predisposing causes* 1 Age Usually 4 months to 4 years 2 Sex Equal incidence 3 Season Winter and spring 4 Locality Central Europe Australia and North America Sporadic cases may occur anywhere Localised epidemics have been described

**Pathology** Peripheral neuritic changes have been described together with chromatolysis of anterior horn cells and at times round celled infiltration of the brain

**Clinical Findings** *Prodromal Stage* The onset is insidious but there is probably an early stage with slight fever lasting a few days The earliest change noted by the parents is often an alteration in their child s disposition The infant may become listless or exhibit fits of temper characterised by screaming and biting He may complain of itching tingling or burning of the soles of the feet or palms of the hands The appetite is often poor and thirst excessive and micturition may be delayed

*On Examination* The child avoids the light and may lie curled up, or bury his head in the pillow or sit forward with his head between his feet The feet and hands are cold and the soles and palms resemble raw beef in their colour and sodden appearance An erythematous rash may also be seen on the trunk and limbs in some cases These skin changes do not always appear and may not be noted until late in the disease Gangrene of the fingers and toes occurs at times The child may be seen rubbing his feet and hands or placing them on a cold surface or sucking his fingers There may be profuse sweating especially of the hands and feet Muscular weakness and hypotonia with wasting may be a prominent feature so that the child cannot walk Myoclonic movements are sometimes seen The teeth may also fall out The deep reflexes are diminished The inguinal axillary and intercostal glands may be enlarged Desquamation of the hands and feet occurs in the terminal stages of the disease before the pink colour disappears The temperature is usually normal but a tachycardia of 140 to 180

persisting by day and night for several weeks is often found The blood pressure is usually raised to 110 to 180 mm Hg The white cells show a leucocytosis of 10,000 to 80,000 per c mm Lumbar puncture in the early febrile stage usually reveals a fluid showing meningitic changes with a slight excess of protein and cells Insomnia may be a very pronounced feature of the disease

**Differential Diagnosis** The early stage is usually considered to be due to some slight febrile disorder The excessive thirst may suggest diabetes insipidus and the muscular weakness poliomyelitis post diphtheritic paralysis or amyotonia congenita Several cases have been mistaken for tuberculous meningitis The gangrene of the fingers or toes may suggest Morvan s disease

**Course and Complications** The disease may last for a few weeks up to six months and may be interrupted by remissions or by exacerba tions and relapses Complications include septicæmia and broncho pneumonia

**Prognosis** This is usually good and there are no sequelæ Pro- gressive wasting or intercurrent infection causes death in about 5% of cases

**Treatment** The child must be very carefully nursed No teething powders should be given A soothing application for the hands and feet consists of a paste made of equal parts of Zinc oxide calcium carbonate, glycerin and water When the burning is very intense the hands can be covered with rubber gloves and placed in ice cold water Some sedative drug, such as phenobarbitone gr $\frac{1}{8}$ for a baby of 4 months and gr $\frac{1}{2}$ for an infant of 4 years should be prescribed for the insomnia Favourable results have been reported by the administration of vitamin $B_1$ This may be given by intramuscular injections of Benerva (aneurin hydrochlor ) mg 2 in 1 ml three times a week In Australia it has been recommended that in mild cases sodium chloride should be given by mouth in doses up to 8 G daily with intramuscular injections of deoxycortone acetate mg 8 on alternate days Good results have been obtained

## Paralysis Agitans

### (*Parkinson s Disease*)

**Definition** A disease characterised by muscular rigidity and tremors It was described by James Parkinson in 1817 in his essay *On the Shaking Palsy* '

**Etiology** The cause is not known *Predisposing causes* 1 Age Usually after 50 2 Sex Males predominate

**Pathology** The lesion is considered to lie in the extrapyramidal motor system of the brain resulting from arteriocapillary fibrosis The corpus striatum, especially the caudate nucleus and the putamen is thought to be affected The corpus striatum probably controls the tone of muscles Lesions of the corpus striatum produce rigidity and tremors resulting from the release of lower centres

**Clinical Findings** The patient first notices some difficulty in per forming actions with one hand his writing may be very small and he

may be conscious of the muscles being somewhat stiff He may also feel an aching pain in the arms or back some general fatigue or perhaps sensations of heat or cold After a variable time the tremors appear

*On Examination* *An early case* Stiffness may be found in the muscles of one hand or forearm and the face may be rather expressionless Slight tremors may be seen in one hand The leg on the same side is often next affected Later the tremors spread to the other hand and the rigidity becomes generalised *A developed case* In the course of about two years the appearance of the patient is very striking The face lacks expression is vacant and somewhat staring and immobile (the Parkinsonian mask) The lower lip may twitch but the eyelids seldom blink The forehead is wrinkled or very smooth The whole attitude is one of flexion the neck and trunk are slightly flexed The shoulders are slightly abducted with the elbows flexed and held away from the body the hands in front of the abdomen The hands are held with the metacarpo-phalangeal joints flexed and the interphalangeal joints extended and the thumb opposed to the index The hips and knees are slightly flexed The movements are all stiff and slow owing to general muscular rigidity On looking to one side the eyes move before the head and the head and trunk move together In walking the gait is shuffling the steps are rather short and the arms swing very little Owing to the flexion of the trunk the balance is easily upset If the patient catches his foot in an object on the floor or if he is pushed from behind he hurries forward with short steps as it were to overtake his centre of gravity This is known as propulsion and the gait is festinant Similarly if the patient is pushed backwards or to one side (retropulsion or lateropulsion) he tends to fall down and to prevent this has to move quickly in the direction in which he has been pushed The tremors have a peculiar rhythmic character of about 4 to 7 vibrations a second They are usually most marked in the fingers The movements of the thumb and index finger produce a pill rolling effect There may also be movements of flexion and extension or radio-ulnar deviation at the wrist or of pronation and supination of the forearm Tremors also occur in the ankles and less frequently in the head and jaw The tremors increase with emotion and can be controlled to a certain degree by voluntary use of the muscles They cease during sleep except in advanced cases It is not uncommon to find that the tremors vary inversely with the degree of muscular rigidity There may also be cramps in the calf with plantar flexion of the toes the big toe being hyperextended Injection of 1% procaine into the motor point of a muscle will abolish the rigidity but the tremors persist The voice is monotonous and it may be high pitched If the patient is watched when he is sitting it may be noticed that he remains quite still apart from the tremors for several hours This is known as poverty of movement Trophic changes may be seen in the skin of the hands and feet which appears smooth glossy red and cold There may be marked sweating at times unilateral There is no evidence of a pyramidal tract lesion thus the deep reflexes

are not increased and the plantar response is flexor Further the posterior columns of the cord and the higher sensory paths are intact as shown by the absence of sensory changes In the later stages the patient becomes bedridden incontinent, and salivation may be troublesome

**Differential Diagnosis** There is little difficulty in recognising a typical case At times the tremor is very slight (paralysis agitans sine tremore), *but there is then definite rigidity and the face is usually* expressionless The aching in the legs may be mistaken for intermittent claudication In senile tremors the musculature is not rigid and the *expression is normal The tremors have not the rhythmicity of paralysis* agitans The Parkinsonian syndrome following encephalitis lethargica can be differentiated by the history of a febrile illness some time previously *This may however have been of a slight nature, and* have passed almost unnoticed The age incidence too is usually much lower, and the rigidity is more marked than are the tremors Salivation *is common in post encephalitic Parkinsonism, and oculogyric crises,* tics and disturbances of sleep may occur A Parkinsonian syndrome may also result from syphilis and poisoning with barbitone manganese *carbon monoxide and carbon dioxide or from cerebral arteriosclerosis* in old people In the latter variety the trunk and legs are chiefly affected and tremors are slight

**Course and Complications** The course *is slowly progressive*, all the voluntary muscles become unduly rigid, and the tremors tend to spread from limb to limb, the patient ultimately becomes bed ridden and is aptly described as a living statue who cannot speak, read or write, but whose intelligence remains clear Complications such as urinary infections bronchitis or bronchopneumonia may develop or a gradual failure of mental function may set in with a terminal coma

**Prognosis** The patient may live for 10 years or more after the onset of symptoms

**Treatment** The patient should be kept warm and in as good a general condition as possible Hyoscine is used to alleviate the tremors It may be given by mouth as a tablet of hyoscine hydrobromide gr 1/200 to gr 1/50 2 or 3 times a day for several years Alternatively hyoscin hydrobrom gr 1/100 may be injected once or twice daily or tnc stramon given in large doses by mouth Fl oz ½ of the tincture produces results comparable with an injection of gr 1/100 of hyoscine If the stramonium produces dryness of the mouth or paralysis of accommodation pilo carpine nitrate gr 1/10 can be added to one or more of the doses daily Various other drugs have been employed

Parpanit is put up in mg 6 25 mg 25 and mg 50 tablets Its main effect is against rigidity The initial dose is mg 12 5 t i d increasing to mg 12 5 q i d then mg 12 5 5 times a day The first daily dose may then be increased to mg 25 and subsequently *the total daily dosage* worked up to mg 125 or more Giddiness weakness and light headedness may sometimes be provoked

Diparcol is also *useful in relieving rigidity It is put up in mg* 50 and mg 250 tablets It may be used in doses of mg 50 t i d the dose

being worked up to a total of 1 G a day *i e* 0 5 G on waking 0 25 G at noon and 0 25 G at 5 p m Side effects include nausea vomiting blurring of vision and drowsiness Agranulocytosis may also occur

Artane is useful in treating rigidity It is put up in mg 2 and mg 5 tablets The dose is mg 2 the first day increased by mg 2 daily up to mg 9 a day divided into three doses If nausea or blurring of vision occurs the drug should be discontinued for some days and then a smaller dose given This drug seems most useful in post encephalitic Parkinsonism

Lysivane has some effect on tremors as well as on rigidity It is put up in mg 50 tablets The dose is mg 50 t i d increased by mg 50 daily to a total of mg 50 10 times a day Side effects include drowsiness vertigo skin irritation dryness of the mouth, and transient diplopia

Benadryl sometimes has a favourable effect It is given in mg 50 capsules 3 or 4 times a day Somnolence can be combated by giving amphetamine sulph mg 5 mane The muscles may be lightly massaged and passive movements employed to relieve rigidity Aching is assuaged by aspirin gr 10 as required Sleep is secured by aspirin gr 10 to 15 or phenobarbitone gr 1 or Soneryl gr 1½ to gr 3 nocte In the terminal stages good nursing is essential

Operation is only advisable for relief of tremor when the disease is strictly unilateral If rigidity and emotional changes are present operation will make matters worse Cordotomy is usually performed in the cervical region with radical division of the lateral pyramidal tract This is followed by hemiplegia which usually recovers in a few weeks

## Progressive Lenticular Degeneration

(*Hepato lenticular Degeneration Wilson s Disease*)

**Definition** A progressive disease characterised by rhythmic tremors muscular rigidity and cirrhosis of the liver

**Etiology** Hepato lenticular degeneration may be due to an abnormal metabolism of copper Radio active copper studies reveal an increased absorption of copper *Predisposing causes* 1 Age Children and young adults The average age is 15 years 2 Familial incidence There is a tendency for the disease to occur in more than one member of a family

**Pathology** Bilateral degeneration occurs in the lenticular nucleus especially in the putamen in which cavities may form The extra pyramidal motor tract the lenticulo rubro spinal tract is presumably deranged Multilobular cirrhosis is seen in the liver The copper content of the brain and liver is increased The pseudoscleroses of Westphal Strumpell and the torsion spasms of Schalbe Ziehen are closely allied conditions

**Clinical Findings** In some cases a history suggestive of a previous disturbance of the liver is obtained such as jaundice with fever and vomiting

*On Examination* Muscular rigidity is found in the limbs and trunk

and bilateral tremors with involuntary movements are seen in the limbs The tremors are increased by voluntary actions but cease during sleep The face may have an emotional expression, such as a spastic smile The patient laughs or cries without adequate reason Greenish brown pigmentation may be seen at the edge of the cornea in some cases There is general muscular weakness and difficulty in balancing Later flexion contractures occur in the arms and legs, with generalised body wasting In the terminal stages the legs may be extended and both speech and swallowing are disturbed There is no alteration in the cutaneous sensation and the deep reflexes are normal The liver is not usually palpable The urine contains from 200 to 700 micrograms of copper in 24 hours derived from the food Normally the urine contains no copper There is also an increased excretion of amino acids in the urine

**Differential Diagnosis** The age incidence serves to distinguish the disease from paralysis agitans and the facial expression also differentiates it from paralysis agitans or the Parkinsonian sequelæ of encephalitis lethargica

**Course and Complications** The course is variable being either acute subacute or chronic, the patient finally becoming bedridden Ascites or hæmatemesis may occur

**Prognosis** The disease is fatal Death occurs in a few weeks to a few years Occasionally the patient may survive for 30 years

**Treatment** There is no cure known It has been suggested that a course of injections of B A L should be given intramuscularly 1 ml of a 10% solution in arachnis oil being injected daily for 20 days This can be repeated every 6 months Calcium disodium versenate mg 500 tab , half a tablet before each meal, diminishes the absorption of copper and so is of value

## Migraine

*(Sick Headache)*

**Definition** Paroxysmal headache usually hemicranial

**Etiology** The cause is not known *Exciting causes* 1 Worry 2 Mental or physical strain 3 Eyestrain 4 Indigestion 5 Menstruation *Predisposing causes* 1 Age The attacks usually begin at puberty, and recur until after middle age 2 Sex Females predominate The attacks are often associated with menstruation 3 Personality Sufferers from migraine are often conscientious hard working perfectionists 4 Heredity In some instances migraine runs in families 5 Other illnesses

**Pathology** Various theories have been proposed to account for migraine They include 1 Cerebral changes The visual disturbances which precede the headache are probably due to occipital cortical vascular spasm The headache is due to dilatation of the extracerebral arteries of the dura and scalp, which are branches of the external carotid artery Raising the cerebro spinal pressure up to 800 mm $H_2O$ does not diminish the headache which indicates that the dural arteries are not affected 2 Allergy 3 Eyestrain 4 Disturbance of liver function

5 Intermittent hydrocephalus of one lateral ventricle due to temporary obstruction of the foramen of Monro 6 Pituitary or ovarian disturbance It is possible that the local vasodilator effect is connected with the presence of certain steroid compounds in the circulation 7 A neurosis

**Clinical Findings** The patient often knows on waking that an attack will develop during the day He has a feeling of malaise with slight headache mental dulness depression yawning cold sensations or giddiness In the course of a few hours further premonitory symptoms may occur such as disturbance of vision The central vision may be blurred or a portion of the visual field cut out. There may be a definite hemianopia A bright spot may appear on the dark visual field or there may be flashes of light or irregular objects with coloured outlines are seen called fortification spectra (teichopsia) In some cases there is a sensory aura with a feeling of numbness or tingling gradually travelling from the hand up the arm usually on the side opposite to that on which the headache develops It may spread to the lips and tongue on both sides The headache gradually develops often on the side opposite to that of the affected visual field The pain of a boring nature often starts in the outer part of one eyeball and spreads over one side of the head and to the upper jaw in the region of the molar teeth It may be definitely throbbing in character and almost intolerable when the patient is standing but relieved by lying The pain may spread to the shoulder Sustained contractions of the muscles of the scalp and neck may occur which are not relieved by ergotamine It is aggravated by movement noises or bright lights Both sides of the head may be affected The patient often feels sick and may finally vomit with relief to his pain In some cases there is a temporary disturbance of speech such as the use of wrong words

*On Examination* The patient usually looks ill and pale but at times the face is flushed The temperature is normal The blood may show an eosinophilia of about 5 to 15% Ophthalmoplegic migraine occurs in some cases with paralysis of the III IV or VI nerves on one side The headache in these cases is usually very intense This may last for a few days or weeks This may be due to an intracranial aneurysm or neoplasm Pressure on the carotid artery on one side may temporarily relieve the headache In other cases a patient who is subject to migraine may experience weakness in one limb or one half of the body on waking which gradually passes off during the day without any headache developing The 17 ketosteroids in the urine are increased during an attack

**Differential Diagnosis** The nature of the headache is usually diagnosed by the hemicrania and periodical recurrence An occipital tumour may give rise to a clinical picture almost indistinguishable from migraine In epilepsy the *sensory aura is of much shorter* duration than that met with in migraine In histamine headache there is dilatation of the cerebral arteries and an attack can be produced by the injection of histamine There is usually no vomiting but it is frequently associated with nasal mucosa congestion

**Course and Complications** The attacks usually last for a day, and the patient does not feel quite normal for another 2 or 3 days They may recur once or twice a week or only at intervals of months Migraine may be followed later in life by epilepsy, or a migrainous subject may beget an epileptic

**Prognosis** The attacks usually cease after the age of 50

**Treatment** In mild cases a dose of aspirin gr 5 to 20 will usually abort the attack In more severe instances work is impossible and the patient has to lie down in a dark and quiet room Some patients obtain relief from phenacetin gr 10 and caffein citrate gr 5 repeated in an hour, if necessary It is better to avoid the regular use of bromides but if the attacks are very frequent and disabling the use of phenobarbitone gr ¼ to 1 every night may hold them in control Ergotamine tartrate owing to its vasoconstrictor effect on the branches of the external carotid artery, will in the majority of cases stop the headache quickly It should not be given in pregnancy or if there is arteriosclerosis It is given as a subcutaneous injection or by mouth Ergotamine tartrate is put up in 1 ml ampoules containing mg 0 5 or as tablets containing mg 1 A test dose of 0 5 ml can be given subcutaneously repeated in 2 hours if headache persists or four mg 1 tablets may be taken by mouth dissolved under the tongue followed by 2 tablets every half hour until a total of mg 10 has been taken If nausea or vomiting is provoked by the ergotamine it can be relieved by a subcutaneous injection of atropin sulph gr 1/100 Ergotamine has no effect upon the frequency of the attacks The patient must lie down for an hour after its administration If the patient does not tolerate ergotamine dihydroergotamine methanesulphonate (D H E 45) may be injected 1 ml (mg 1) and repeated once or twice at half hourly intervals Nitroglycerin has a reputation for diminishing the frequency of the attacks and may be given as the tabella glyceryl trinitrat gr 1/120 t d s or as Gowers mixture Liq trinitrin m 1 liq strychnin hydrochlor m 5 sod brom gr 10 acid hydrobrom dil m 10 tinc gelsemii m 5 aq chlorof ad fl oz ½ Fl oz ½ ex aqua t d s Between the attacks the patient should sedulously avoid constipation fats should be restricted in the majority of cases and any ocular refractive error or muscular imbalance should be corrected

### Headache

Recurrent headache is a very common minor malady, although in some cases it proves extremely disabling and in others is a symptom of grave disease The following account is largely based on the writing of H G Wolff (Clinics 1944 2 1394)

**Etiology** Intracranial causes of headache include 1 Traction on veins passing to the venous sinuses or displacement of the sinuses 2 Traction on the middle meningeal arteries 3 Traction on the larger arteries at the base of the brain 4 Distention and dilatation of the intracranial arteries 5 Inflammation around pain sensitive structures of the head 6 Pressure by tumours on cranial and cervical nerves

Pain may be referred to the head in angina pectoris

Varieties of Headache 1 *Due to changes in intracranial pressure* either above or below the normal Removal of 20 ml of spinal fluid with the patient in the erect position will cause headache which can be relieved by the intrathecal injection of saline This is called a *traction headache* Bilateral compression of the jugular veins will increase headache On the other hand increasing the intracranial pressure to a level of 550 mm $H_2O$ does not itself cause headache which indicates that increased intracranial pressure does not of itself cause headache

2 *Brain tumour headache* This is usually intermittent in early cases It is deep seated dull and not throbbing It is worse on coughing and is not usually so severe as the headache of migraine or of meningitis It is produced chiefly by traction on arteries veins venous sinuses or nerves It is almost always present with posterior fossa tumours It is located over the site of the tumour in about one third of the cases It is rarely occipital in supratentorial tumours unless there is papill œdema When it is both occipital and frontal it is a sign of extensive cerebral displacement

3 *Histamine headache* This is due to dilatation and stretching of pial and dural arteries It can be produced by the intravenous or subcutaneous injection of histamine The headache does not occur immediately after the injection when the arterial relaxation is maximal but it is most marked when the blood pressure returns to its initial level The large arteries at the base of the brain are chiefly responsible for this type of headache It may occur in people with increased histamine sensitivity and throbbing may be felt in arteries in other parts of the body such as the chest wall Pressure on the trunk as by lying in bed or against a chair may cause release of histamine

4 *Fever headache* There is increased pulsation of the cerebral arteries Increasing the pressure of the fluid in the subarachnoid space lessens the headache

5 *Migraine* This has been described above

6 *Headache associated with high blood pressure* This is due to dilatation and distention of the branches of the external carotid artery as in migraine In some cases it is due to hypertensive encephalopathy In cases of high blood pressure strain and fatigue may cause relaxation of the arteries and thus produce headache The headache is not relieved by increasing the cerebro spinal fluid pressure Ergotamine tartrate which acts chiefly on the branches of the external carotid artery relieves the headache Pressure on the temporal frontal supraorbital posterior auricular and occipital arteries diminishes the headache

7 *Nasal and paranasal and mastoid headaches* These are associated with sinus or mastoid disease In sphenoidal and ethmoidal sinusitis the pain is felt between the eyes at the back of the eyes and over the vertex Frontal sinus headache often begins early in the morning and goes on all day with maxillary antral disease the headache tends to begin in the afternoon The headache is non pulsatile

8 *Ocular headache* This may be due to errors of refraction errors of muscle balance or glaucoma Myopia does not cause headache because the myope does not contract his eye muscles as this would make his

vision worse Electromyograms show abnormalities of the scalp muscles in ocular headaches

9 *Cervical headache* This variety of pain is known as *tension head ache* Tender spots may be felt in the occipital muscles Anxiety is a common cause The patient may have pain, a feeling of pressure or paræsthesia on the vertex Contraction of the scalp, head and neck muscles is often present In some cases the headache is due to loss of the normal lordotic curvature of the cervical spine due to a sprain of the paravertebral ligaments

This type of headache is best treated by rest in bed massage and heat applied to the neck The patient should be reassured there is no serious disease, such as a cerebral tumour and aspirin gr 10 should be given 3 or 4 times a day, together with phenobarbitone gr ½ morning and evening In some cases stretching of the neck muscles is beneficial

10 *Headache due to general diseases* The most important amongst these are chronic nephritis anæmia and syphilis Strychnine may be a cause of headache

**Clinical Notes** Certain clinical observations are of value in diagnos ing the variety of headache The most common causes are migraine and emotional tension The most severe headache is due to subarachnoid hæmorrhage and meningitis Intracerebral hæmorrhage rarely causes headache unless the blood escapes into the ventricles or subarachnoid space Headache usually becomes severe in the terminal phases of cerebral tumour before the stage of coma Pulsating headaches are met with in migraine fever high blood pressure, histamine sensitivity and hæmangiomatous tumours A continuous ache is characteristic of a cerebral tumour in the later stages A feeling of a tight band round the head or of a tight cap is due to muscle spasm associated with eye causes sinus infection and emotions If the headache is always on the same side there may be an aneurysm of the circle of Willis A bitemporal headache is met with in pituitary adenoma

### *Obsessive-Compulsive Neurosis*

*(Tics Habit Spasms)*

**Definition** Repetitive purposive movements originally performed in response to a mental or physical stimulus which tend to persist after the exciting cause has been removed

**Etiology** In a few cases an external physical cause has existed such as conjunctivitis or a frayed stiff collar In the majority of instances however no such cause can be traced There is a neuropathic tendency which leads to the perpetuation of the tic *Predisposing causes* 1 Age The tic often starts about puberty, but it may begin earlier or later 2 Sex The incidence is equal

**Pathology** No organic lesion of the nervous system can be found A tic may be regarded as a conditioned reflex the stimulus being an associated psychical one

**Clinical Findings** Certain varieties of tic are described such as 1 *Simple tic* Here only individual muscles or a few muscles are

involved Thus the patient blinks frequently when talking or twitches his mouth or eyebrow He may constantly turn his head shrug his shoulder cough or sniff The same tic is repeated at short intervals and is likely to be more pronounced if the patient is nervous 2 *Co ordinated and convulsive tics* The movements are more complicated thus the patient may repeatedly make stooping movements as he walks along or he may have attacks of convulsive movements accompanied by explosive words such as swearing (coprolalia) or words or actions may be repeated or copied (echolalia and echokinesis) 3 *Psychical tics* The patient is the victim of various obsessions Thus when walking he feels he must touch each lamp-post he passes, and if he misses one he goes back and touches it

**Differential Diagnosis** The brisk nature of the repetitive muscular contractions usually renders the diagnosis of a tic clear In some cases the movements which occur in chorea focal epilepsy torsion spasm or in encephalitis lethargica may require consideration

**Course and Complications** Tics usually continue without change A psychical tic may prove so distressing as ultimately to lead to insanity or suicide

**Prognosis** The chance of a tic disappearing is less if it has persisted for a long time or if it begins after middle age

**Treatment** Any reflex source of irritation should be removed The general health should be improved as far as possible by proper exercise and a sufficiency of fresh air and food Parents should not direct attention to the tic as although this may lead to the disappearance of one tic it is usually attended by the development of a fresh one An endeavour should be made 2 or 3 times a day to relax the muscles affected the patient lying down and concentrating his attention on keeping the muscles still Further exercises should be performed in which the affected muscles are contracted and relaxed systematically In some cases hypnotic suggestion has been attended with success

## Professional Cramp

*(Occupational Neurosis)*

**Definition** Fatigue and cramp affecting groups of muscles employed in skilled occupations

**Etiology** The cramp occurs as the result of prolonged use of certain muscles associated with a neuropathic tendency

**Pathology** The fatigue process is believed to occur not in the muscles or in the peripheral nerves but centrally in the brain

**Clinical Findings** Many varieties are described such as writer s cramp musician s telegraphist s typist s haircutter s and cigarette-maker s cramp The patient is usually a male between the ages of 20 and 50 and a skilled worker who is performing his specialised task In the early stages there is aching or stiffness in the muscles and if the worker persists he may have to stop owing either to definite weakness or cramp of the muscles After a short rest and rubbing the muscles he is able to resume If however he endeavours to persist in his

occupation, the disability becomes more marked and finally it is impossible for him to carry on

**Differential Diagnosis** Every case should be carefully examined to exclude such lesions as peripheral neuritis tenosynovitis or an early stage of paralysis agitans The diagnosis is usually clear, as certain groups of muscles are affected and only when a definite act is performed

**Course and Complications** The course is progressive as described above

**Prognosis** The outlook is unfavourable unless adequate treatment is given, and even then relapse is liable to occur

**Treatment** A rest of at least 6 months from the provocative occupation is required In writer s cramp the patient should learn to use a large penholder he should not grip it tightly, and he should write from the shoulder rather than from the fingers and wrists

## Hysteria

**Definition** A condition characterised by mental dissociation which may lead to somatic symptoms sensory disturbances to personality changes or amnesia without any organic disease of the nervous system being present

**Etiology** The true cause of hysteria remains unknown It is believed by some that there is a restriction of the fields of consciousness and that the symptoms develop so that the patient may escape from the realities of mental emotions or physical hardships They result therefore either from auto- or hetero suggestion The Freudian school consider that hysteria is due to a repression of conflicts usually of a sexual nature Hysteria may follow a severe mental or physical shock or be due to suggestion especially in epidemics of hysteria It may be purposive a method of avoiding an unpleasant duty or a reaction to neglect or mental suffering *Predisposing causes* 1 Age Usually between 15 and 35 years 2 Sex Females predominate 3 Heredity A neuropathic tendency is an important factor 4 Race In Europe the Teutonic races are comparatively immune the Latins Slavs and Jews being susceptible 5 Class Hysteria is more common in the hospital class of patient

**Pathology** No organic lesion is found in the brain

**Clinical Findings** The patient is usually an adult woman lacking emotional control and introspective She is not worried about her symptoms and may be mentally dull The clinical picture varies according to the type of hysteria present

1 *Motor Types* (a) *Convulsive hysteria* This occurs in attacks and is sometimes known as hystero-epilepsy *Minor attacks* The patient may have preliminary disturbances such as palpitations the feeling of a lump rising in the throat (globus hystericus) or distressed breathing She then falls in the presence of onlookers but is careful not to injure herself Various clonic muscular contractions take place, but the patient is not really unconscious she does not bite her tongue or micturate, and the corneal reflexes persist *Major attacks (grande*

*hystérie*) This is very uncommon in England but was met with chiefly in France at the end of the last century The attack may last for several hours The early stages are characterised by clonic and tonic muscular contractions The patient may scream and assume various contortional and emotional attitudes On recovery visual and auditory hallucinations may persist for a time The attacks occur in the presence of others, and probably are largely due to suggestion Somnambulism is considered to be a manifestation of major hysteria (*b*) *Non-convulsive motor hysteria* The patient may complain of inability to move an arm or leg or the arm and leg on the same side of the body or both legs

*On Examination* Various types of motor hysteria may be seen such as a monoplegia hemiplegia or paraplegia Further there may be contractures irregular movements or catalepsy In the hysterical paralysis certain distinguishing features can usually be detected. Thus a patient may say she cannot stand or walk (astasia abasia) and yet she can move her legs perfectly when lying down The muscles do not waste the electrical reactions are normal and the deep reflexes are often exaggerated The plantar response is flexor in type If a patient is asked to bend a paralysed arm the antagonistic muscles can often be felt to contract opposing any flexion result which would be produced by contraction of the agonists In a hysterical paralysis of a leg if the patient is asked to raise her body when lying with her arms across her chest the sound leg will come up in the air but the paralysed one remains on the bed This is due to muscular contraction in the paralysed leg and is the reverse of that which takes place in an organic monoplegia where the paralysed leg rises higher than the sound one Further in hysterical monoplegia sensory cutaneous loss of a hysterical type is generally present The gait differs from that of an organic lesion in that the ' paralysed leg tends to be drawn along after the body and is not swung outwards as in an organic lesion The adductor muscles of the vocal cords may be affected *Spasmodic contractions* Various muscles of the trunk limbs or face may be involved Thus there may be trismus or ptosis due to contracture of the orbicularis palpebrarum without compensatory wrinkling of the forehead The contraction does not usually cease during sleep but is abolished by anæsthesia The phantom abdominal tumour is of this nature there being spasms of the abdominal muscles in localised areas with some flatulent distension *Irregular movements* These consist of tremors muscular twitchings and chorei form or repetitive movements Various complicated actions such as bowing may be repeatedly performed There may be attacks of laugh ing or crying *In catalepsy* the patient goes into a trance the limbs are rigid and remain in whatever position they are placed

2 *Sensory Types* The patient may complain of every imaginable type of pain There may be headache like a nail being driven into the skull (clavus hystericus) defective vision such as mistiness inability to bear a bright light or blindness in one eye The patient may complain of deafness or increased sensitivity to sounds Other symptoms include deficiency of taste (often bilateral) or of smell a lump in the throat loss of appetite flatulent dyspepsia constipation

retention of urine, yawning hiccough coughing inability to take a full breath, palpitations, sweating or flushing *Psychical types* include cases of dual personality and amnesia The patient is reported as "missing and probably suffering from loss of memory"

*On Examination* In hysterical blindness of one eye, the patient may be seen to blink when a blow is directed at the affected eye, the other being covered In hysterical deafness, the patient may be awakened by a noise of average intensity Certain hysterical stigmata may be found the most important being cutaneous and pharyngeal anæsthesia and restriction of the visual fields The patient is unaware of these stigmata and some authorities maintain they result from suggestion on the part of the examiner This is doubtful, they are more probably due to auto suggestion *Cutaneous anæsthesia* The patient does not injure the anæsthetic part There may be hemianæsthesia, or a sock, stocking, glove or sleeve anæsthesia in a limb The upper limits of anæsthesia are fairly sharply defined Joint sense is unaffected The patient may really feel when the skin is stimulated as is shown by asking her to say "yes" when the skin is pricked and 'no" when it is not pricked She may fall into the trap and say "no" when the pin is applied to the "anæsthetic" area A stimulus applied to one limb may be referred to a corresponding spot on the other limb (allocheiria) Areas of hyper æsthesia may be found on the chest or abdomen and so called hysterogenous spots These are tender spots, especially likely to be present in the left inguinal region Pressure on them may induce a hysterical fit *Reflexes* The conjunctival abdominal and plantar reflexes may be lost in cases of cutaneous anæsthesia The pharyngeal reflex is not necessarily lost in hysteria if care is taken to avoid suggestion *Restriction of the visual fields* The patient does not complain of loss of vision in any direction before perimetric tests are carried out The spiral restriction of the visual field, as determined by the perimeter appears to be the result of suggestion

**Differential Diagnosis** Convulsive hysteria is differentiated from epilepsy by the points mentioned on p 347 In many cases the diagnosis of hysteria presents no difficulties, when the characteristic findings described above are considered It should always be remembered that a hysterical element may be implanted on an organic lesion and may persist when the organic lesion is healed or arrested In every case a complete examination should be carried out not once only, but at intervals of several weeks In this manner the early stages of an organic disease such as disseminated sclerosis will not be over looked In malingering there is a deliberate attempt to deceive, which is not the case in hysteria

**Course and Complications** Hysterical symptoms may persist for many years or disappear suddenly at any time There is always a tendency for the cure to be temporary and for subsequent recurrence

**Prognosis** Miraculous" cures may result from suggestion, the blind recovering his sight or the mute his power of speech

**Treatment** In some cases a convulsive attack may be arrested by firm pressure on a so-called hysterofrenic spot such as the supra

orbital notch The most hopeful method of cure is by persuasion The physician must acquire the complete confidence of his patient who is made to believe that a cure is possible and will be effected The nature of the complaint is explained to the patient In some cases the use of electrical stimulation is of value in abolishing cutaneous anæsthesia or in demonstrating to the patient that his muscles will contract

## Neurasthenia

### (*Anxiety States*)

**Definition** A condition characterised by abnormal fatigue of the mind or body without a discoverable organic cause

**Etiology** No definite cause can be assigned to neurasthenia it is sometimes considered to be a fatigue neurosis Pavlov has produced experimental neurosis in dogs as the result of overtaxing inhibition processes by conditioned reflexes *Exciting causes* 1 Overwork 2 Worry 3 Illness such as influenza anæmia or enteric fever 4 Drugs such as alcohol or cocaine 5 Injury producing traumatic neurasthenia *Predisposing causes* 1 An inherited neuropathic tendency 2 Age Usually between 20 and 45 years 3 Sex Males predominate

The Freudian school believes that neurasthenia is due to some sexual trauma which may only be made manifest when one of the exciting causes is operative

**Clinical Findings** The patient is usually an adult of lugubrious expression and with many complaints although in some cases he looks extremely fit In order to avoid forgetting any symptom he may bring a list of them written out The examiner usually finds that the list is not easily exhausted and when asked if there is anything else that he has noticed wrong the patient will produce another complaint In neurasthenia bodily complaints predominate whereas in psychasthenia the symptoms are chiefly mental The onset is usually gradual the patient feeling tired and lacking interest in anything except himself The fatigue is abnormal the patient wakes feeling tired but often improves as the day goes on The symptoms in neurasthenia may be referred to the various systems of the body The patient may complain of loss of appetite flatulent dyspepsia or of occipital headache He often states that there is a feeling of oppression on the top of the head or of constriction of the head The skull may feel empty or too full He may suffer from palpitations dizziness flushing sweating a frequent desire to yawn a sensation of a lump in the throat numbness or tingling in various parts of the body Backache may be marked and sexual disturbances such as impotence a prominent symptom In psychasthenia the patient may have groundless fears Certain of these have been granted names such as fear of open spaces (agoraphobia) of closed spaces (claustrophobia) of disease (pathophobia) of crowds (anthropophobia) of solitude (monophobia) and of dirt (mysophobia) The patient feels unable to concentrate to read or to keep still He has needless worries and suffers from insomnia He may be the victim of various

obsessions a prey to drink (dipsomania) to drugs (toxicomania) or to stealing (kleptomania)

*On Examination* The patient often looks anxious, but the nutrition is usually good The peripheral circulation may be poor and the hands cold and bluish There are no signs of organic disease discoverable but the deep reflexes are brisk In the cases of flatulent dyspepsia the patient may make loud eructations the whole time that he is being examined The blood pressure in neurasthenia is usually low, and the pulse rate unstable varying more than 10 beats in the standing and recumbent position Traumatic neurasthenia follows some accidental injury, such as a railway accident when often no external injury is visible Railway spine may then develop with pain in the back and weakness, although no organic lesion is discoverable by X ray or other examination

**Differential Diagnosis** Every care should be taken to exclude organic disease before a diagnosis of neurasthenia is made Thus early pulmonary tuberculosis is at times diagnosed as neurasthenia a mistake which would not occur if the morning and evening temperatures were taken, and the chest carefully examined physically and radiographically A cerebral tumour in a silent area of the brain may also cause disturbance of health with vague sensations of distress and pain without any localising signs being found Later, with increasing intracranial pressure the diagnosis becomes apparent General paralysis of the insane at its onset may be mistaken for neurasthenia If there is doubt, the blood and cerebro spinal fluid should be examined An early case of Addison's disease may also be diagnosed as neurasthenia, a blood pressure as low as 100 mm Hg systolic should suggest the former diagnosis

***Course and Complications*** *Neurasthenia is a chronic condition,* which is liable to persist unless adequately treated Insanity is a rare complication

**Prognosis** The outlook is fairly good but, despite treatment, the neuropathic disposition remains and relapses are not *infrequent*

**Treatment** The most important factor in successful treatment is the discovery and elimination of the exciting cause whether it is due to physical strain, mental worry, toxæmia or trauma In severe cases a complete rest change of environment, and a congenial occupation are required Apart from this the treatment is general such as extra diet in the form of a glass of milk or cup of cocoa in the morning some exercise daily out of doors and early hours at night If there is difficulty in sleeping a dose of phenobarbitone gr 1 to 2 should be given at night In traumatic neurasthenia an improvement is often noted when the responsibility for treatment is definitely cast upon the patient by settlement of any compensation claim

## THE CEREBELLUM

### Anatomy and Physiology

The *cerebellum* is composed of three parts a central vermis and two lateral lobes The vermis contains the following nuclei The

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## THE CEREBELLUM

### Anatomy and Physiology

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nucleus fastigii (roof nucleus) and the nucleus globosus on each side Each lateral lobe contains the dentate nucleus and the nucleus emboliformis The cerebellum is connected with the brain by three peduncles on each side the inferior peduncle or restiform body the middle peduncle or brachium pontis and the superior peduncle or brachium conjunctivum (see Fig 31)

Developmentally the cerebellum consists of two parts separated by the posterolateral fissure These are the flocculonodular lobe with afferent and efferent vestibular connections equilibratory in function and the corpus cerebelli which integrates proprioceptive impulses from muscles The corpus cerebelli is divided by the fissura prima into an anterior and posterior lobe The anterior lobe consists anatomically of the lingula lobus centralis and culmen Physiologically the lobulus simplex belongs to the anterior lobe The anterior lobe controls postural

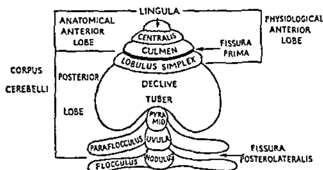



FIG 99 DIAGRAM OF THE CEREBELLAR CORTEX SHOWING PRINCIPAL DIVISIONS (After Dow and Fulton)

reflexes and the synergies of walking The posterior lobe is formed anatomically of the lobulus simplex declive tuber pyramid and uvule This lobe controls ipsilateral skilled voluntary movements

The paleocerebellum consists of the flocculonodular lobe the anterior lobe of the corpus cerebelli the uvule and pyramid The neocerebellum is formed by the greater part of the hemispheres and the vermis (see Fig 32) Ablation of the nodulus in dogs affords complete protection against motion sickness

**The Efferent Tracts** There are three main efferent tracts from the cerebellum

1 *The Cerebello rubro spinal Tract* From the cortex of the lateral lobe through the superior peduncle to the opposite red nucleus The second relay of fibres conducts the impulses down the rubro spinal tract which crosses at its origin so that it runs down to the anterior horn cells on the same side of the body as that from which the impulses arose in the cerebellum The final relay is by the anterior nerve roots to the skeletal muscles

2 *The Cerebello cerebral Tract* From the cerebellar cortex to the dentate nucleus where the second relay passes through the superior cerebellar peduncle to the thalamus of the opposite side The final relay conducts the impulses to the frontal and post central cerebral cortex

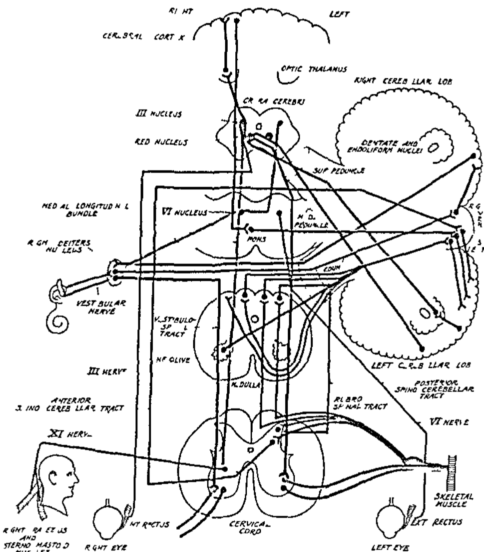



Fig. 31 Diagram of the Cerebellar Connections

2 *Primary Progressive Degeneration* (Sanger Brown ataxia see p 438) The fibres running from the cortex to the nuclei are affected

3 *Olivo-ponto-cerebellar Atrophy* There is atrophy of the cortex middle peduncles and part of the inferior peduncles The inferior olives are also affected

4 *Vascular Lesions* Thrombosis is liable to affect the posterior inferior cerebellar artery Hæmorrhage may occur from the superior cerebellar artery

5 *Tumours* These include a glioma endothelioma sarcoma psammoma lipoma myxoma and cholesteatoma The medullo blastoma of childhood arises in the flocculonodular lobe in embryonal rests It is a relatively common tumour The cystic angioma of the cerebellum may be associated with an angiomatous retinal tumour (v Hippel s disease) Multiple angiomatous tumours may also occur in the cord kidney liver, spleen and pancreas (Lindau s disease) Degenerative cysts also occur they are generally gliomatous or sarcomatous

6 *Granulomata* A tuberculoma or gumma may be found

7 *Abscess* This may be secondary to suppuration in the mastoid or labyrinth, or to spread of infection from lateral sinus thrombosis The infection may be blood borne as in bronchiectasis or infective endocarditis In some cases the abscess results from a wound of the cerebellum which may have been inflicted some time before

8 *Disseminated Sclerosis*

9 *Cortical Cerebellar Atrophy*

The most commonly occurring lesions are a glioma and a tuber culoma

**Clinical Findings** The results of cerebellar lesions vary according to whether they are irritative or paralytic and also whether the lateral hemisphere or vermis is affected Neighbouring structures may also be involved such as the cranial nerves especially the VIII VI and V and the pons with disturbances of either the motor or sensory tracts *Irritative lesions* These result usually from a wound or hæmorrhage *Paralytic lesions* These may be due to thrombosis a tumour or an abscess

## Cerebellar Hæmorrhage

The patient complains of very severe vertigo He feels as if his body is being rotated away from the side of the lesion and as if sur rounding objects are also rotating in the same direction There may also be pain in the occipital region with vomiting and the patient often falls to the ground his body being rotated on its long axis by forced movements

*On Examination* The patient is often found lying with the side of his face corresponding with the site of the lesion on the pillow Spontaneous nystagmus may be seen with slow movements away from the side of the lesion and sharp short movements in the opposite direction There may also be skew deviation of the eyes the eye on the affected side looking downwards and inwards and the other eye

8 *The Cerebello Deiters' Tract* From the cerebellar cortex thr the inferior peduncle to Deiters' nucleus *of the same and opposite s* Relays of fibres connect thence with the vestibulo spinal tract run down to the anterior horn cells on the same side, and so by the relay to the skeletal muscles and by the medial longitudinal bu with the III, IV and VI nuclei regulating eye movements and with nuclei of the spinal portion of the XI nerve controlling head movem

The Afferent Tracts There are six main tracts

1 *The Direct Cerebellar or Dorsal Spino cerebellar Tract of Flecl* This runs through the inferior peduncle to the vermis on the same s The final relay carries the impulses to the cerebellar cortex *of the lat* lobe of the same side

2 *The Indirect Cerebellar Tract or Ventral Spino cerebellar Trac Gowers* This runs up the opposite side of the cord and ascends to level of the red nucleus It turns down and crosses the mid line to superior cerebellar peduncle and so passes to the vermis The fi relay carries the impulses to the cerebellar cortex of the lateral lobe the side at which the impulses entered the spinal cord

3 *Fibres arising from the Cuneate and Gracile Nuclei* These form superficial and deep arcuate fibres and pass through the infe peduncle to the vermis The final relay is to the cortex of the late lobe of the cerebellum, on the same side as that on which the major of impulses entered the cord

4 *The Olivary cerebellar Tract* Fibres pass from the inferior ol *in the medulla cross the mid line, and enter the cerebellum by* inferior peduncle They relay in the vermis, the final path being to cerebellar cortex of the lateral lobe on the opposite side

5 *The Deitero cerebellar Tract* Fibres run from Deiters nucl through the inferior peduncle to the homolateral nucleus fastigii of vermis A second relay conveys the impulses to the cerebellar cort of the lateral lobe of the same side

6 *The Cerebro cerebellar Tract* Fibres pass from the frontal, te poral and occipital lobes to the pons A second relay conveys t impulses by the middle peduncle to the vermis The final relay runs the cerebellar cortex of the opposite lobe

*The cerebellum is thus intimately connected* with the skeletal muscl on both sides with the impulses from Deiters' nucleus concerned wi equilibrium and with the co ordination of the eye and head movemen

*The chief function of the cerebellum is* to enforce and control t postural tone of the skeletal muscles which is of vital importance f synergic or co ordinated movements The hemispheres are chief concerned with the tone of muscles on the same side of the body, ar the vermis with the backward or forward balance of the body and wi the conjugate deviation of the head and eyes *phonation and articulatio*

## Lesions of the Cerebellum

The following lesions may occur 1 *Hypoplasia* This is a congenit lesion The cerebellum may be partly or completely absent

the patient tending to walk round it as if it were a crutch The arm on the affected side often remains motionless *Attitude* The occiput may be pointing towards the shoulder on the affected side which is usually slightly lower than and in front of the opposite shoulder there is some scoliosis with concavity towards the side of the lesion

5 *The Rebound Phenomenon* The patient holds his arms flexed and the examiner pulls against them On releasing the pull on the arm on the affected side the patient's hand flies back to hit his face or body whereas this movement is automatically checked on the sound side

6 *Dysdiadokokinesis* The patient is unable to perform rapid alternate actions on the affected side such as pronation and supination of the forearm or closing and unclosing the hand

7 *Nystagmus* This occurs typically as a fixation nystagmus which is not present when the eyes are at rest If the patient is asked to look to the affected side nystagmus appears the eyes slowly swinging away from the fixation position owing to lack of tone in the muscles and being rapidly jerked back again If the patient looks to the sound side the nystagmus consists of smaller movements which are more rapid and irregular but there is a slow movement away from the fixation position and a rapid jerk back With bilateral lesions nystagmus occurs on looking to either side the slow movement being away from the fixation point and the quick movement towards it Vertical nystagmus may occur with lesions of the vermis Nystagmus may also be provoked by caloric stimulation of the ears Further if the stimulation produces conjugate deviation of the eyes the lesion is also probably central There is also often difficulty in looking towards the side of the lesion

8 *Vertigo* This may persist the patient feeling that both he and the surrounding objects are rotating away from the side of the lesion

9 *The Knee-jerks* The knee on the side of the lesion shows a pendulum swing due to lack of tone in the extensors

10 *Tremors* Intention tremor may occur in the homolateral limbs If the arms are held out static tremors may be seen on the affected side the hand dropping a little and then being jerked upwards

11 *Cerebellar Catalepsy* This term is applied to the abnormal steadiness which may at first be seen in the homolateral arm or leg when it is held out before the static tremor develops

## Cerebellar Thrombosis

Clinical Findings Thrombosis of the posterior inferior cerebellar artery is known as the Wallenberg syndrome There is usually a comparatively sudden onset with ataxia on the affected side but the patient does not lose consciousness In addition owing to involvement of the medulla there may be dissociated anæsthesia (loss of pain and temperature sensation but not of touch) of the trunk and limbs on the opposite side with homolateral dissociated anæsthesia of the face and paralysis of the palate and vocal cord Vomiting dysphagia and Horner s syndrome on the affected side may also be noted Anti

upwards and outwards In many cases a hæmorrhage involves the lateral region of the medulla When the spino thalamic tract is affected there is analgesia for pain and temperature sensations on the same side of the face and head and on the opposite side of the body and limbs Involvement of the nucleus ambiguus may result in interference with swallowing and speech An irritative lesion may cause a cerebellar fit, characterised by a sudden onset, with loss of consciousness and tonic spasm The head is retracted and the back arched The elbows, hips knees and ankles are extended The patient does not usually bite his tongue or micturate With unilateral lesions the homolateral leg is adducted and the heterolateral leg abducted The patient rotates to the side of the lesion and the eyes deviate to the opposite side

### The Cerebellar syndrome

Special signs and symptoms are described which constitute the "cerebellar syndrome" These signs cannot be detected in every case and are obscured if the lesion presses on the medulla and interferes with the pyramidal tracts The chief features of the cerebellar syndrome are as follows 1 *Hypotonia* The muscles of the body on the same side as the lesion are affected This may be demonstrated when the patient is lying down by the extent to which the patella can be depressed on the two sides when the muscles are relaxed The leg can also be abducted farther on the affected side The arm is more flail like if the forearm is shaken up and down by grasping the upper arm In the past pointing test the arm on the affected side deviates away from the object

2 *Asthenia* The grip is less powerful on the affected side and the limb will tire suddenly and completely

3 *Ataxia* This can be shown in various ways Thus there is asynergia or decomposition of movements The muscles do not work together smoothly in performing such an action as putting the foot on a chair Astasia consists in jerky clonic contractions of muscles, and can be tested by feeling the biceps while the patient flexes his forearm with his elbow resting on a table Dysmetria (disturbance of the range of movement) Movements are often performed excessively (hyper metria) Thus there is a tendency to overshoot the mark in the finger nose test and in taking up a glass the hand opens too widely when seizing it and when putting it down There may also be delay in relaxation of muscles so that the glass is knocked over when the patient takes his hand away from it Associated movements Irregular clonic movements may occur in the homolateral limb when forced muscular contractions are made in the limb on the sound side In some cases there is a tendency on standing to fall backwards or to one side Cerebellar ataxia is not increased on closing the eyes Ataxia of speech occurs especially in vermis lesions the speech being either staccato or drunken

4 *Gait* This is reeling or drunken the patient tending to deviate or fall to the affected side There is difficulty in standing on the leg on the affected side On walking also the affected leg swings outwards

side in the direction of the lesion Optic neuritis is frequently marked Muscle hypotonus may be detected on the affected side and dysdiadokokinesis may be easily recognised in the arms

**Differential Diagnosis** A cerebellar abscess is often hard to diagnose and may be overlooked It may give rise to no localising signs If it closely follows a purulent labyrinthitis no fresh signs or symptoms may be noted Lumbar puncture serves to differentiate septic meningitis If nystagmus persists after operation for purulent labyrinthitis there is probably also a cerebellar abscess

**Course and Complications** The course is usually progressive Pyæmia and purulent meningitis may occur as complications

**Prognosis** Death usually occurs in from 1 to 2 months unless the abscess can be satisfactorily drained

**Treatment** This is surgical as soon as the abscess has been diagnosed it should be drained

### Cortical Cerebellar Atrophy

The ganglion cells of the cerebellar cortex may undergo atrophy in late middle age possibly due to arteriosclerotic changes The gait becomes ataxic and later the arms are affected with inco ordination and intention tremors There is usually no nystagmus but the plantar responses may be extensor A subacute form has also been described associated with primary carcinoma usually in the lungs or in the ovary Diplopia is frequently present and dementia may rapidly ensue

## THE CRANIAL NERVES

### I The Olfactory Nerve

**Anatomy** The olfactory nerves receive impulses of smell from the back of the nose The fibres pass through the cribriform plate of the ethmoid bone and travel back to the olfactory bulb and olfactory tract which lies in a groove on the orbital surface of the frontal lobe The olfactory tract divides centrally into two roots the medial crosses to end in the uncinate gyrus of the opposite side and the lateral connects with the uncinate gyrus of the same side of the brain

**Lesions of the Olfactory Nerve** The nerve endings may be affected in acute or chronic rhinitis The nerve fibres may be damaged by a fracture of the base of the skull by pressure of a tumour of the frontal lobe olfactory groove or pituitary region or by basal meningitis Atrophy of the nerve may occur in tabes dorsalis Toxins such as alcohol and nicotine and infections such as influenza may result in loss of smell

**Clinical Findings** The sensation of smell is tested in each nostril by asking the patient to inhale such substances as oil of cloves pepper mint lemon or asafœtida Anosmia implies loss of smell and in such cases food appears tasteless In hyperosmia the patient is unduly sensitive to smells This may be a manifestation of hysteria or result from drugs such as strychnine or cocaine Parosmia is a state of per verted sensation of smell a pleasant odour being considered repugnant

coagulants may be given as described on p 187 for 4 or 5 days with good results

### Cerebellar Tumours

**Pathology** Benign tumours in children include the astrocytoma and in adults the hæmangioblastoma The medulloblastoma is the common malignant tumour

**Clinical Findings** The patient is often a child or young adult who complains of headache, and later disturbance of vision vomiting and vertigo may occur

*On Examination* The generalised increased cranial pressure accounts for the headache, vomiting and optic neuritis The localised cerebellar lesion gives rise to a variable number of the signs described above as constituting the cerebellar syndrome Extension of the tumour may cause paralysis of the VI nerve or by pressure on the pons hemiplegia on the opposite side If a tumour is situated in the vermis there is a tendency for the patient to fall backwards or forwards The medullo blastoma of the flocculonodular lobe in children causes unsteadiness of gait accompanied by walking on a wide base

### Extra cerebellar Tumours

*(Ponto cerebellar Angle Tumours)*

**Pathology** A meningioma arises from the meninges covering the under surface of the cerebellum and a neurofibroma from the sheath of a cranial nerve, generally the vestibular part of the VIII nerve

**Clinical Findings** The patient is usually an adult who first complains of deafness and tinnitus in one ear Later there is headache and vertigo the patient feeling that he is rotating to the side of the lesion but that objects are rotating away from the side of the lesion

*On Examination* Nerve deafness is found on the affected side and there may be signs of involvement of the VI or VII nerves or the sensory part of the V nerve Diminution or loss of the corneal reflex may be an early sign The pyramidal tract on the opposite side may be affected by pressure of the medulla against the skull There is then a spastic hemiplegia on the same side as the tumour Symptoms of a lateral cerebellar lesion may also appear, but these will be largely masked if the pyramidal tract is involved

### Cerebellar Abscess

**Clinical Findings** The patient complains of headache occipital or frontal and there may be vertigo and stiffness of the neck and some drowsiness A history is sometimes given of recent mastoid or labyrinthine trouble with deafness or tinnitus

*On Examination* The patient may be apathetic the temperature is often raised although sometimes it is normal Spontaneous nystagmus may be seen the short quick movement being towards the side of the abscess The patient may lie on the sound side with the knees flexed The finger nose test often shows deviation of the arm on the affected

side in the direction of the lesion Optic neuritis is frequently marked Muscle hypotonus may be detected on the affected side and dysdiadokokinesis may be easily recognised in the arms.

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or *vice versa* It occurs at times in association with a tumour or with empyema of the antrum In epilepsy an olfactory aura may precede an attack

## II The Optic Nerve

Anatomy (see Fig 33) There are two visual fields, one for each eye, and each may be divided into an *inner or nasal and an outer or* temporal part The retina of each eye may also be divided into two parts the inner which receives impressions of objects in the temporal field of vision, and the outer, which is the receptor for the nasal visual field The optic nerve passes from the retina and meeting its fellow in the mid line intermingles to form the optic chiasma from which the optic tracts proceed back to connect with the primary visual centres The fibres from the nasal halves of the retinæ decussate in the optic chiasma and pass to the primary visual centres on the opposite side of the brain, whereas the fibres from the temporal halves of the retinæ are carried on in the homolateral optic tract The macular fibres from each retina undergo a partial decussation some fibres crossing in the chiasma and others passing back in the optic tract of the same side The primary visual centres are the external geniculate body, the superior corpus quadrigeminum and the pulvinar of the optic thalamus It is very doubtful, however whether the optic thalamus is a visual centre From the external geniculate body relays of fibres convey the visual impulses to the cerebral cortex of the occipital lobe especially to those con volutions situated around the calcarine fissure in the area striata' on the mesial surface of the occipital lobe The fibres first pass through the posterior limb of the internal capsule and then run back in the optic radiation to the occipital cortex Owing to the crossing of the macular fibres in the optic chiasma, the right half of each macula has its cortical centre in the right occipital lobe close to the occipital pole and the left half of the macula in the left occipital lobe Fibres also run from the occipital lobe to the optic *thalamus and the superior corpus quadri* geminum The first relay of fibres in the optic tract which ends in the superior corpus quadrigeminum is situated close to the nucleus of origin of the III cranial nerve on the opposite side and a reflex arc for light impulses is thus established between the retina and the ocular muscles Connections are also established with the IV and VI nuclei The III nerve in addition to conveying fibres supplying certain extrinsic eye muscles is also the channel for impulses passing to the intrinsic ocular muscles Thus the cranial autonomic fibres run in the III nerve to the ciliary ganglion where a fresh relay starts the short ciliary nerves supplying the ciliary muscle and the constrictor of the pupil The dilator muscle of the pupil is supplied by the sympathetic chain which passes to the superior cervical ganglion A fresh relay conducts the impulses through the Gasserian ganglion of the V nerve to the ophthalmic branch of this nerve and the impulses travel along the long ciliary nerves to the dilator muscle *With these anatomical facts* clearly established the effects of lesions at various points in the visual path are easily appreciated (see Fig 33)

FIG 33 DIAGRAM OF THE VISUAL PATH

*Lesion at* 1 (optic nerve) = Blindness of eye
*Lesion at* 2 (centre optic chiasma) = Bitemporal hemianopia
*Lesion at* 3 (outer side chiasma) = Nasal hemianopia
*Lesion at* 4 (optic tract) = Bilateral homonymous hemianopia with abolition of light pupil reflex
*Lesion at* 5 (optic radiation) = Bilateral homonymous hemianopia with light pupil reflex normal
*Lesion at* 6 (calcarine cortex) = Bilateral hemianopia (peripheral vision)
*Lesion at* 7 (occipital pole) = Loss of central vision left halves of maculæ

[T face p 376.

**Lesions of the Optic Nerve and Visual Path** The most important affections of the optic nerve are neuritis and atrophy

### Optic Neuritis, Papillitis and Papillœdema

**Definition** These terms are applied to inflammation and swelling of the head of the optic nerve within the globe of the eye

**Etiology** Optic neuritis is usually bilateral and may be due to increased intracranial pressure resulting in increased tension of fluid in the nerve sheath with compression of veins or it may be a manifestation of neuro retinitis The most common cause of increased intracranial pressure is a tumour of the cerebrum or cerebellum Other causes include cerebral abscess meningitis especially when due to tuberculosis and chronic hydrocephalus Neuro-retinitis may be due to nephritis diabetes mellitus syphilis or leukæmia Unilateral optic neuritis results from orbital periostitis inflammation of the orbital connective tissue a retro-orbital tumour localised meningitis herpes zoster affecting the cornea or an aneurysm of the ophthalmic or internal carotid artery

**Clinical Findings** The patient may complain of headache but the vision may be apparently normal, although definite papillœdema is present Later attacks of blurred vision or definite impairment of sight occur especially of central vision so that reading is impossible

*On Examination* In the early stages the pupils are normal but later they are dilated and do not react to light Ophthalmoscopic examination shows the disc red and swollen with a blurred edge The retinal veins are dilated and the arteries are small In papillœdema or choked disc there is much œdema and swelling of the disc In addition in neuro-retinitis retinal changes such as patches of exudate and hæmorrhages are seen

**Prognosis** This depends upon the cause of the neuritis When due to syphilis there is usually a good response to treatment

**Treatment** A decompression operation should be performed in cases due to cerebral tumour in order to endeavour to save the sight before optic atrophy develops

### Retro bulbar Neuritis

**Definition** Inflammation of the optic nerve behind the eye

**Etiology** Retro bulbar neuritis may be due to 1 Disseminated sclerosis syphilis affecting the optic nerve or a hæmorrhage into the sheath of the optic nerve 2 Sepsis in the ethmoidal sphenoidal or frontal sinuses in the cranial antra and possibly in other sites of the body such as the teeth 3 Vitamin B deficiency 4 Neuromyelitis optica (Devic s disease) 5 Toxic causes especially tobacco alcohol methyl alcohol and tryparsamide 6 Metabolic disturbances such as diabetes mellitus and possibly rheumatism Exposure to cold is a predisposing cause

**Clinical Findings** When retro bulbar neuritis develops acutely it is usually unilateral The patient complains of progressive loss of vision and often there is pain on moving the eye

*On Examination* Pressure on the eyeball may cause pain In the early stage the pupil appears normal but it may be found that on exposure to a bright light the pupil first contracts but subsequently dilates while still exposed to the light The disc is usually normal but there may be slight redness and blurring of the disc margin Determination of the visual fields usually reveals a central scotoma (blind spot) where either white objects or certain coloured objects are not seen This is due to a lesion of the papillo macular bundle and is called axial neuritis The peripheral visual field may alone be restricted in interstitial peripheral neuritis In more advanced cases sight is completely lost (diffuse neuritis) and there is optic atrophy with pallor of the optic disc

**Prognosis** This is on the whole favourable but relapses may occur and both eyes may be affected

**Treatment** This varies with the cause The patient should stop smoking and take no alcohol A shade should be worn over the affected eye Septic foci should be searched for and removed Syphilis or diabetes mellitus, if present must be adequately treated (see p 596) If no specific cause is found iodides should be given in increasing doses with sodium salicylate such as Pot iod gr 5 to 30 sod salicyl gr 5, sod bicarb gr 10 syr aurant m 20 aquam ad fl oz 1 Fl oz 1 t d s p c

## Primary Optic Atrophy

**Definition** Atrophy of the optic nerve not preceded by a stage of optic neuritis The distinction between primary and secondary optic atrophy is artificial and based on ophthalmoscopic findings In all cases of primary atrophy there is some cause such as pressure on the nerve central to the entry of the vessels or a toxin affecting the nerve

**Etiology** The atrophy may be due to 1 Nervous diseases such as tabes dorsalis general paralysis of the insane amaurotic family idiocy, a frontal lobe tumour pressure on the optic chiasma or nerve due to a pituitary tumour and possibly disseminated sclerosis 2 Toxic substances such as lead arsenic in the form of Atoxyl or tryparsamide or methylated spirit 3 A hereditary variety, known as Leber s disease 4 Severe hæmorrhage or anæmia 5 Glaucoma and retinitis pigmentosa

**Clinical Findings** The patient complains of progressive loss of sight One or both eyes may be affected

*On Examination* The pupil is dilated and does not react to light The disc is white with a sharp margin the surrounding retina being normal The field of vision diminishes from the periphery, without a central scotoma

**Prognosis** This is unfavourable

**Treatment** This is unsatisfactory If the optic atrophy is due to tabes dorsalis or general paralysis of the insane the treatment detailed on p 407 should be given

## Secondary Optic Atrophy

**Definition** Degeneration of the optic nerve subsequent to optic neuritis

**Etiology** The causes are similar to those mentioned on p 377 for optic neuritis, such as a cerebral tumour aneurysm or hydrocephalus

**Clinical Findings** The patient complains of loss of sight.

*On Examination* The disc is pale but the edges are blurred and the retinal arteries thread like

**Prognosis** This is very unfavourable

**Treatment** No treatment is likely to be efficacious when the stage of atrophy has been reached

## The Optic Chiasma, Optic Tract, Optic Radiations and Calcarine Region

Lesions affecting these portions of the visual path are usually due to tumours vascular disturbances meningitis or aneurysm of the internal carotid artery

**Clinical Findings** The fields of vision are mapped out with the aid of a perimeter Normally the visual field is most extensive for white objects the fields for blue red and green objects being smaller in the order of colours given The visual fields may be found cut off in definite directions Thus in bitemporal hemianopia both temporal fields are blind This results from a lesion affecting the central part of the chiasma (see Fig 83) In the early stages the upper temporal quadrant of one visual field is usually first affected by tumours of the pituitary which cause pressure from below upwards whereas in suprasellar tumours with pressure from above downwards the early loss is likely to occur in the lower temporal quadrant In nasal hemianopia the nasal visual fields are eliminated This is a rare condition due to a lesion affecting the outer part of the chiasma on both sides It may be due to atheroma of the internal carotid arteries to distension of the third ventricles due to an intracranial tumour or to chronic internal hydrocephalus causing pressure of the outer part of the chiasma against the internal carotid arteries In bilateral homonymous hemianopia the nasal field of one eye and the temporal field of the other are affected This results from a lesion of the optic tract or radiation on one side A pituitary tumour may cause such a lesion In quadrant hemianopia one quadrant of the visual field is affected such as the upper half of one temporal and the upper half of the other nasal field or the lower half of one temporal and the lower half of the other nasal field This results from a lesion around the calcarine fissure the superior quadrant of the visual field being represented below the calcarine fissure and the inferior quadrant of the visual field above the calcarine fissure When the occipital pole is affected there may be central blindness half the macula on each side being represented on each occipital pole (see Fig 83) Lesions of the occipital cortex may result in mind blindness (optic agnosia) the patient sees objects but cannot recognise them Visual hallucinations may also occur Wernicke s hemiopic pupillary reaction is described as an aid to determine whether a lesion of the visual path is situated before or behind the primary visual centre of the superior corpus quadrigeminum A narrow beam of light is cast

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into the eye by an ophthalmoscope mirror on to the blind half of the retina If the pupil contracts the light reflex fibres are not involved and so the lesion is situated behind the primary visual centre, *i e*, in the internal capsule, optic radiation or cortex In practice it is very difficult to carry out this test

*Amaurosis* is a term used to indicate blindness *Uræmic amaurosis* may occur in the course of acute or chronic nephritis The patient suddenly becomes blind in one or both eyes The pupils are dilated but react to light The discs and fundi are normal apart from any changes associated with the nephritis There is usually spontaneous recovery in the course of a day or so (see p 487)

*Amblyopia* indicates some dimness of vision In hysterical amblyopia the pupils and fundi are normal but the fields of vision show a spiral restriction during testing either from within outwards, or from without inwards, according to the manner in which the test is conducted Hemianopia may also occur in hysteria or in migraine

## The III, IV, and VI Nerves

### (*The Oculo motor Nerves*)

*Anatomy* The III nucleus lies in the mid brain at the level of the superior corpora quadrigemina in the floor of the Sylvian aqueduct Some fibres cross joining the III nerve of the opposite side The nerve

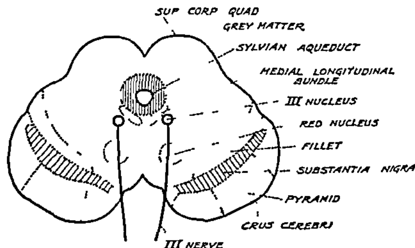



FIG 34 DIAGRAM OF THE MID BRAIN AT THE LEVEL OF THE SUPERIOR CORPORA QUADRIGEMINA

leaves the brain on the ventral surface near the mid line on the inner side of the crus and passes through the outer wall of the cavernous sinus to enter the orbit through the sphenoidal fissure (see Fig 34) It divides into a superior branch supplying the levator palpebræ superioris and the superior rectus muscles and an inferior branch distributed to the inferior and internal recti and the inferior oblique muscles It also gives fibres to the ciliary ganglion from which the short ciliary nerves

run to the ciliary muscle and the constrictor of the pupil The IV nucleus is situated in the mid brain at the level of the inferior corpora quadrigemina in the floor of the Sylvian aqueduct The IV nerve decussates in the valve of Vieussens and leaves the brain on the opposite side on the dorsal surface (see Fig 35) It passes forwards around the crus cerebri and runs in the outer wall of the cavernous sinus below the III nerve Passing through the sphenoidal fissure it ends in the superior oblique muscle The VI nucleus is situated close to the VII nucleus in the lower part of the pons in the floor of the fourth ventricle It leaves the brain on its ventral surface at the lower border of the pons and runs in the inner wall of the cavernous sinus just external to the internal carotid artery It passes through the sphenoidal fissure to the orbit and supplies the external rectus muscle The ophthalmic branch of the V nerve also runs in the outer wall of the cavernous sinus the nerves

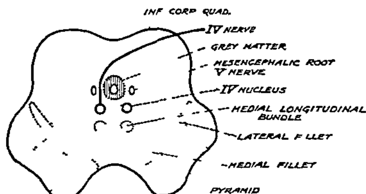



FIG 35 DIAGRAM OF THE MID BRAIN AT THE LEVEL OF THE INFERIOR CORPORA QUADRIGEMINA

being in the following order from above downwards III IV ophthalmic branch of V and VI (see Fig 28)

**The Oculo motor Muscles** The external muscles of the eye produce movements of the eyeball in three directions 1 Vertical an upwards or downwards movement 2 Horizontal an inwards or outwards movement 3 Torsion or wheel motion the vertical meridian being rotated inwards or outwards

The movements effected by the muscles are as follows *The external rectus muscle* This moves the eye outwards *The internal rectus muscle* This moves the eye inwards *The superior rectus muscle* This moves the eye upwards and inwards and rotates or twists it inwards *The inferior rectus muscle* This moves the eye downwards and inwards and twists it outwards *The superior oblique muscle* This moves the eye downwards and outwards and twists it inwards *The inferior oblique muscle* This moves the eye upwards and outwards and twists it outwards *Diplopia* or double vision results from paralysis of the external ocular muscles It results from the axes of the eyeballs not being parallel when an object is looked at The patient therefore

sees two objects as the images do not fall on corresponding parts of the retinæ The true image is the one seen by the sound eye It is more distinct than the false image which is seen by the affected eye A red glass is put before the affected eye and an object such as a candle is looked at the false image then appears red and the true image white In homonymous or simple diplopia the false image appears on the side of the true image corresponding with the side of the lesion, *e g* if the

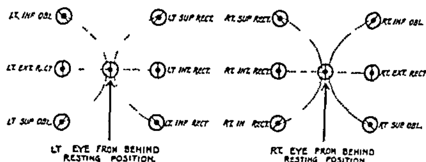



Fig 36 Diagram of the Action of the Oculo-motor Muscles (After Swanzy and Werner)

left eye is affected then the false image is to the left of the true image, or *vice versâ* This is the case always in convergent (internal) strabismus In crossed diplopia the false image is situated on the opposite side of the true image This occurs in divergent (external) strabismus The action produced by the various oculo motor muscles can be seen from the accompanying diagram (see Fig 36)

The movements upwards downwards inwards and outwards and

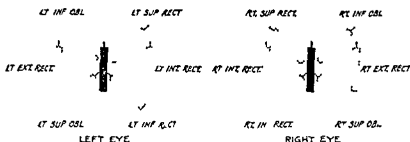



Fig 37 Diagram of False Images in Oculo motor Palsies (After Swanzy and Werner)

the torsion movements of the eyeball produced by the various muscles are all represented Thus the left inferior oblique moves the eyeball upwards and outwards and rotates or twists it outwards The position of the false images can also be conveniently represented by a similar diagram the false image being featured by the dotted line drawing of the candle (see Fig 37) The false image is always situated in the direction of pull of the paralysed muscle

*Paralysis of the superior rectus muscle* There is weakness in moving the eye upwards and inwards The diplopia is present on looking upwards it is crossed the false image being above and tilted

away from the true image *Paralysis of the inferior rectus muscle* There is weakness in moving the eye downwards and inwards The diplopia is present on looking downwards it is crossed the false image being below and tilted towards the true image *Paralysis of the external rectus* There is inability to move the eye outwards The diplopia is homonymous the false image is on the same level as the true one and on the same side of it as is the lesion Thus with a left external rectus paralysis the patient complains of diplopia when he looks to the left There is an internal strabismus the affected eye being turned inwards by the unopposed action of the internal rectus The head may be kept a little turned to the affected side Secondary deviation may be detected in the sound eye Thus if the patient is asked to look outwards with the affected eye and the sound eye is screened the sound eye moves inwards to a greater degree than the affected eye moves outwards This is because a greater stimulus than normal is given to the affected eye to try and cause it to move and a correspondingly greater contraction takes place in the internal rectus of the sound eye which normally works in unison with the external rectus of the other eye The farther the test object is moved to the affected side in determining the diplopia the greater is the distance between the true and false images The patient also misjudges distances on looking to the affected side, the object appearing farther away than it really is *Paralysis of the internal rectus* There is inability to move the eye to the affected side There is external strabismus The patient has crossed diplopia on looking inwards the false image being on the opposite side but on the same level as the true one *Paralysis of the inferior oblique* There is weakness in moving the eye upwards and outwards and in rotating outwards There is diplopia on looking upwards The patient may elevate the head and then look downwards to avert the diplopia which is homonymous the false image being situated above and tilted away from the true image *Paralysis of the superior oblique* There is weakness in moving the eye downwards and outwards and in rotating it inwards The patient complains of diplopia and giddiness on looking down as on descending a flight of stairs The diplopia is homonymous the false image is below and tilted towards the true image

**Lesions of the Oculo motor Nerves** Lesions may occur in three portions of the motor tract from the cerebral cortex to the muscles of the eye 1 Supranuclear lesions in the cortex corona radiata internal capsule or mid brain 2 Nuclear affecting the III IV or VI nuclei 3 Infranuclear affecting the nerves themselves

### Supranuclear Lesions

**Etiology** These may result from vascular disturbances such as hæmorrhage thrombosis or embolus or from the pressure of a tumour The cortical centre for eye movements is thought to be situated in the region of the second frontal gyrus

**Clinical Findings** Both eyes are affected There is usually a disturbance of conjugated lateral movements In bilateral internal capsular lesions or in unilateral mid brain lesions in the region of the

superior corpora quadrigemina or Sylvian aqueduct there may be disturbance of conjugated vertical movements of both eyes There is usually an associated hemiplegia Thus with a right sided *lesion there* is paralysis of the left side of the body, with conjugate deviation of the eyes The direction of this deviation depends upon whether the lesion is an irritative or a paralytic one With an irritative lesion the eyes look away from the lesion *i e*, if the lesion is on the right side of the brain the eyes are deviated to the left The reverse holds good with a paralytic lesion This is explained by reference to the diagram (see Fig 88) The cortical lesion affects the VI nucleus on the opposite side, and the VI nucleus is connected to, and works synergically with the portion of the III nucleus of the opposite side which supplies the internal rectus muscle

Treatment This varies with the cause, as for cerebral vascular *lesions or tumours*

## Nuclear Lesions

**Etiology** The oculo-motor nuclei may be stimulated or paralysed by such lesions as a hæmorrhage, thrombosis or embolus a mid brain or pontine tumour A cerebellar tumour may affect the VI nerve nucleus by indirect pressure Other lesions include those produced by acute encephalitis occurring as a complication of measles, influenza scarlet fever or small pox encephalitis lethargica, tabes dorsalis, syringomyelia, chronic poliomyelitis disseminated sclerosis Wernicke s

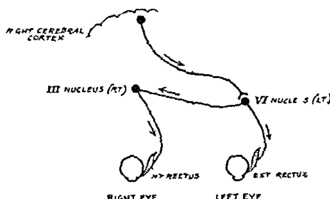



FIG 88 DIAGRAM OF THE MECHANISM OF CONJUGATE DEVIATION OF THE EYES

encephalopathy (see p 648) and botulism A nuclear lesion may also occur in myasthenia gravis

**Clinical Findings** Nuclear ophthalmoplegia may appear as an acute or chronic disease In the acute variety there is often headache and vomiting with a raised temperature Both eyes are affected usually groups of muscles but not all the muscles are involved There may also be internal ophthalmoplegia the pupil being moderately dilated, and showing no response to light or to accommodation usually however, the intraocular muscles are not affected An irritative nuclear

lesion causes a conjugate deviation of the eyes so that they look towards the side of the lesion and a paralytic lesion has the converse effect This is the reverse of what occurs in a supranuclear lesion and is explained by the diagram (see Fig 38) If the pyramidal tract is affected there is hemiplegia on the side of the body opposite to the lesion (Weber s syndrome) If the rubro spinal tract is also involved there are involuntary tremors of the opposite side of the body Chronic nuclear paralysis is more often met with The onset of the symptoms is insidious there is usually conjugate deviation of the eyes the patient not being able to turn the eyes in one lateral direction It is often difficult to differentiate between single nuclear lesions and infra nuclear lesions when only one muscle is affected If in addition to some of the muscles supplied by the III nerve the orbicularis palpebrarum muscle is also paralysed the lesion is probably a nuclear one Lesions of the VI nucleus are usually accompanied by a VII nerve nuclear lesion as well A VI left nuclear lesion causes conjugate paralysis to the left side but if the left eye is covered the right eye can be moved inwards and both eyes will converge

*Jaw winking* may occur in cases of congenital ptosis When the jaw is depressed to the opposite side by contraction of the external pterygoid muscle of the same side the impulse passing along the V nerve causes the upper lid on the same side to be twitched up the paralysed levator palpebræ superioris contracting It is usually unilateral

## Infranuclear Lesions

**Etiology** The oculo motor nerves may be affected at different parts of their course 1 *In the brain* The lesions here are usually vascular or due to tumours Increased intracranial pressure associated either with supra or infratentorial growths by stretching the nerve may cause VI nerve paralysis This constitutes a false localising sign of a VI nerve lesion 2 *Between the pons and the orbit* The lesion may be meningitis often syphilitic a gumma an intracranial tumour, neuritis due to diphtheria, diabetes mellitus or less often to lead alcohol or nicotine toxic neuritis from septic foci elsewhere fractured base of the skull cavernous sinus thrombosis aneurysm or atheroma of the internal carotid or posterior communicating artery and subarachnoid hæmorrhage The VI and V nerves may be affected in acute mastoiditis in children probably due to a localised meningitis *In Gradenigo s syndrome* there is weakness of the external rectus muscle on one side due to involvement of the VI nerve by inflammation of the apex of the petrous bone in association with otitis media The patient is ill with fever and complains of a headache on the same side Ophthalmo plegic migraine may be due to unilateral distension of the lateral ventricle *The sphenoidal fissure syndrome* Acute periostitis may cause infranuclear paralysis a condition comparable with peripheral facial palsy There is then pain in the eye with partial or total ophthal moplegia and proptosis The ophthalmic and maxillary divisions of the V nerve may also be involved with cutaneous anæsthesia over their area

of distribution Overgrowth of bone as in leontiasis ossea may cause pressure paralysis Other causes of the syndrome are aneurysm of the internal carotid artery and meningeal tumour In these cases proptosis is liable to occur The nerves are likely to be involved in the following order VI IV ophthalmic division of V, III and maxillary division of V It then results in ophthalmoplegia, anæsthesia of the eye, cheek and half the forehead with severe pain 3 *In the orbit* The nerves may be involved in retro orbital tumours or inflammation of the connective tissue behind the eye 4 *The terminations of the nerve in the ciliary muscle and constrictor of the pupil* The vegetable mydriatic alkaloids atropine hyoscyamus and hyoscine, cause paralysis by affecting the myoneural junctions

**Clinical Findings** One or more of the III, IV and VI nerves may be involved

*The Third Nerve* All the muscles supplied by the nerve or only some of them may be paralysed With a complete third nerve paralysis there is ptosis with compensatory contraction of the occipito frontalis muscle as shown by wrinkling of the forehead The eyeball is deviated outwards by contraction of the external rectus muscle It cannot be turned inwards or upwards and only slightly downwards The superior oblique muscle may cause by its action a slight downward and outward movement in a wheel like manner The pupil is moderately dilated and does not contract to light or to accommodation The results of partial III nerve paralysis have been considered above under paralyses of the various muscles *Internal ophthalmoplegia* The ciliary muscle and the constrictor and the dilator of the iris may be paralysed The pupil is usually of medium size and does not react to light or to accom modation Internal ophthalmoplegia may be due to a lesion of a portion of the III nucleus or of the fibres passing to the ciliary ganglion or of the Gasserian ganglion In post diphtheritic paralysis the ophthalmoplegia may be unilateral or bilateral

## The V Nerve

### (*Trigeminal Nerve*)

**Anatomy** The V nerve leaves the brain at the outer part of the middle of the pons by a large sensory and a small motor root The motor nucleus lies in the upper part of the pons under the lateral part of the floor of the fourth ventricle The mesencephalic nucleus and mesen cephalic root extend from the upper part of the mid brain to the level of the motor nucleus It is usually considered to be motor in function Upper motor neurones pass from the cerebral cortex on each side to connect with these motor nuclei Thence lower motor neurones pass out from the pons in the V nerve Some authorities believe that the mesencephalic nucleus is composed of cells analogous to the Gasserian ganglion and posterior root ganglia of the spinal nerves which have migrated into the brain and convey proprioceptive impulses from the masticatory muscles The Gasserian ganglion contains the cells of origin of the sensory fibres which enter the pons Some of the fibres conveying kinæsthetic sensations from the three divisions of the V nerve

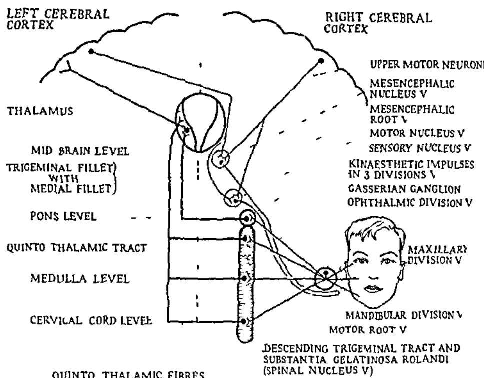



Fig 39 Diagram of the Central Path of the V Nerve

[To face p 38

pass to the sensory nucleus relay there cross the mid line and ascend as the trigeminal fillet to relay in the thalamus Thence the impulses pass to the cerebral cortex Others, conveying pain and temperature sensations run down the pons medulla and cord to the level of the 2nd or 3rd cervical segment as the spinal or 'ascending' root ending at different levels in the spinal nucleus in the substantia gelatinosa Rolandi These fibres form the descending trigeminal tract

The second relay from the substantia gelatinosa passes to the opposite side as the quinto thalamic fibres and ascend as the quinto thalamic tract to end in the lateral nucleus of the thalamus The sensory fibres in the mandibular, maxillary and ophthalmic divisions, conveying pain and temperature sensations decussate in the Gasserian ganglion so that they end in the spinal nucleus in this order from above downwards inversely to their distribution to the face (see Fig 39)

The V nerve after leaving the pons passes forwards over the petrous bone where the Gasserian ganglion is situated on the sensory part of the nerve the motor part lying beneath this ganglion Three main nerves emerge from the Gasserian ganglion named the ophthalmic or first the maxillary or second and the mandibular or third division The motor nerve is attached to the third division *The ophthalmic branch* runs in the outer wall of the cavernous sinus and passing through the sphenoidal fissure to the orbit terminates in the lachrymal frontal and nasal nerves In the cavernous sinus it gives a branch to the dura mater The lachrymal nerve supplies the lachrymal gland the conjunctiva cornea and the outer canthus of the eye The frontal nerve divides into the supra orbital and supra trochlear nerves and supplies the skin on the front half of the scalp and forehead the root of the nose and inner canthus of the eye the upper eyelid and the frontal sinus The nasal nerve supplies the mucous membrane of the upper part of the nose and the skin of the lower part and tip of the nose

*The maxillary branch* runs along the lower part of the cavernous sinus enters the foramen rotundum and spheno maxillary fossa and traverses the orbit to appear on the face through the infra orbital foramen In the skull it gives a recurrent branch to the dura mater In the infra orbital canal it supplies branches to the teeth of the upper jaw Its cutaneous branches are distributed to the face between the lower eyelid side of the nose and upper lip Branches are also given to the mucous membrane of the hard palate and the antrum of Highmore *The mandibular branch* leaves the skull through the foramen ovale and gives a recurrent branch to the dura mater The motor fibres supply the following muscles The masseter the temporal the external and internal pterygoids the mylohyoid the anterior belly of the digastric the tensor tympani and the tensor palati The auriculo temporal branch supplies cutaneous branches to the temple and scalp and to the external auditory meatus and upper part of the pinna This nerve also gives branches to the parotid gland and the temporo mandibular joint The lingual branch supplies the tactile filiform papillæ in the anterior two-thirds of the tongue The chorda tympani nerve joins the lingual nerve and conveys taste sensations from the tongue to the VII nerve

and then to the glosso pharyngeal nucleus The *inferior dental branch* supplies the teeth of the lower jaw, and emerging through the mental foramen innervates the skin over the chin and lower lip The mucous *membrane of the mouth, gums and Eustachian tube* is also supplied by this division of the V nerve

**Lesions of V Nerve** The following varieties of lesions may occur 1 *Supranuclear Lesions* The cortical centres for the muscles innervated by the V nerve are situated in the lower third of the precentral convolution These centres are bilateral each centre sending fibres to both V nuclei and so both centres must be put out of action to cause an upper motor neurone paralysis of the masticatory muscles Lesions may occur here in the corona radiata, internal capsule or mid brain They are rare and include meningeal and vascular disturbances and tumours In epilepsy, hydrophobia and post encephalitic syndromes *supranuclear disturbances of the V nerve may occur In paralysis* agitans there is probably a lesion in the corpora striata disturbing the fibres in the internal capsule In strychnine poisoning and tetanus the lesion is probably situated in the synapses connecting the upper motor neurones with the nuclear cells

2 *Nuclear Lesions* These are situated in the pons and include hæmorrhage, tumour gumma, polioencephalitis disseminated sclerosis bulbar paralysis amyotrophic lateral sclerosis and syringomyelia

3 *Infranuclear Lesions* The V nerve may be affected at the base of the brain by meningitis or fracture of the base of the skull, or its branches may be involved by a tumour thrombosis of the cavernous sinus an aneurysm of the internal carotid artery and cellulitis of the orbit The Gasserian ganglion may be inflamed, or the branches of the V nerve involved in a neuritis, such as occurs in diphtheria

**Clinical Findings** *Disturbances of the V nerve may affect* chiefly the motor or sensory fibres or combined lesions may occur 1 *Motor Lesions* These may be irritative or paralytic (*a*) *Irritative lesions* *Tonic or clonic contractions of the masticatory muscles occur* The former are known as trismus and may be present in tetanus epilepsy tetany or strychnine poisoning Clonic spasms are seen with rigors and paralysis agitans, etc Nocturnal jaw grinding is a *functional* nervous disturbance and thought to be an indication of mental protest (*b*) *Paralytic lesions* Supranuclear lesions when bilateral are characterised by paralysis of the lower jaw The patient can neither close the mouth, protrude the jaw nor move it from side to side Food tends to drop from the mouth There is no wasting of the muscles and no reaction of degeneration Nuclear lesions are often bilateral with a flaccid paralysis of the masticatory muscles on both sides Infranuclear lesions are usually unilateral There is often wasting and weakness of the muscles of mastication on one side of the face When the patient opens his mouth the lower jaw is pushed to the paralysed side by the action of the opposite external pterygoid muscle This is best judged by the position of the central incisors of the upper jaw when the mouth is open and closed There is inability to move the jaw laterally to the sound side, and on opening the mouth the condylar process of the

lower jaw can be felt not to move forwards on the affected side When the patient is asked to bite it can be felt that the masseter and temporal muscles do not contract, and some flaccidity of the floor of the mouth may be found on pressing below the chin The jaw jerk is absent and the affected muscles show a reaction of degeneration

2 *Sensory Lesions* (*a*) *Loss of Sensation* An upper motor neurone lesion affecting the posterior part of the internal capsule may cause loss of sensation of the face the arm body and leg on the opposite side of the body (crossed hemianæsthesia) A lesion of the spinal root of the V nerve and the mesial fillet on the same side will cause alternate hemianæsthesia the face on one side and the arm body and leg on the other side being affected Lower motor neurone lesions may affect individual branches of the V nerve Thus in periostitis of the orbit the ophthalmic division may be affected with certain oculomotor nerves as described on p 385 There is then blunting of sensation in the cutaneous area of this nerve around the orbit and forehead and the corneal and conjunctival reflexes may be abolished with diminished tear secretion When the maxillary and mandibular branches are involved in addition to the cutaneous anæsthesia the patient cannot feel his tongue and cheek normally and is apt to bite it and in drinking from a glass he can only feel half the glass touching his lip There may also be diminished secretion of saliva on one side the tongue is dry and furred on the affected side and using a tuning fork of low pitch there may be loss of appreciation for low notes Anæsthesia is found in the cheek and gums Smell may be diminished on one side owing to dryness of the nasal mucous membrane Taste is not affected unless the chorda tympani nerve is also involved Segmental blunting or loss of sensation may also occur limited to trigeminal dermatomes around the mouth The teeth on the affected side may fall out Facial or corneal herpes and corneal ulceration may also be a manifestation of lesions of the sensory division of the V nerve or of the Gasserian ganglion

(*b*) *Trigeminal Neuralgia* This may be of a minor degree with pain situated in the region of certain branches of V nerve Thus there may be supra orbital neuralgia associated with frontal sinusitis Tenderness may be found at the supra orbital notch Headache may be due to involvement of the branches supplying the dura mater The severe type of trigeminal neuralgia is known as *tic douloureux* This is an affection of middle age or later but it is occasionally met with in children There are periodical paroxysms of agonising pain in the face especially in the areas supplied by the second and third divisions of the V nerve The pain may spread to the neck A paroxysm lasts for a few seconds or minutes and tends to recur frequently During the attack the face may become flushed and moist with perspiration and there may be watering of the eyes and mouth and twitching of the facial muscles The attack may be provoked by the slightest stimulus such as a draught washing or shaving cleaning the teeth or eating

*On Examination* Tender spots may be found over the infra orbital or mental foramina

**Treatment** In all cases of trigeminal neuralgia the cranial sinuses should be investigated frontal sinusitis may result in supra orbital or *supra trochlear neuralgia* The pain is best relieved by local heat which can be conveniently applied by a rubber hot water bottle A nasal spray should also be used containing some preparation such as the Chloretone Inhalant Dental extraction rarely relieves the pain and should not be advised unless an obviously diseased tooth is present In *tic douloureux* the patient should have complete rest in bed for several weeks and for the pain the following mixture may be given Tinc gelsemii m 10, sod salicyl gr 5 sod bicarb gr 20, sod brom gr 10 syr aurant m 20 aquam ad fl oz 1 Fl oz 1 t d s p c Good results have been reported by the daily subcutaneous injection of 1 000 micro grams of vitamin $B_1$ for 10 to 14 days, followed by a similar dose twice a week for several months In severe and protracted cases relief may in some cases be obtained for several months by injecting the Gasserian ganglion or its branches with m 10 to 15 of 80% alcohol through the foramen ovale or foramen rotundum This is an operation which should only be performed by a specialist It may be repeated later if the pain recurs In very severe cases ganglionectomy or resection of the posterior nerve root inside the skull may be necessary to relieve the pain Even then the patient may still feel pain although the sensory fibres are cut off from the brain This is comparable with the sensation which may be felt in the toes after a leg has been amputated

3 *Trophic Lesions* It has been mentioned above that corneal ulceration may follow lesions of the first division of the V nerve If the eye is protected by a shade from the irritation of dust etc these lesions do not occur They are probably mechanically produced the irritants not being removed by blinking as they are not felt Facial hemiatrophy is a peculiar condition in which there is wasting in structures supplied by the V nerve on one side The cause is not known but it may be due to any nuclear or infranuclear trophic disturbance of the V nerve, and is at times associated with osteitis of the jaw or it may follow erysipelas The bones cartilages and soft tissues are affected chiefly in the areas supplied by the second and third divisions of the V nerve One side of the face is smaller than the other The skin becomes thin and the subcutaneous fat disappears there may also be atrophy of half of the tongue There are no sensory changes the muscles are not paralysed, and there is no reaction of degeneration

*The auriculo temporal syndrome* consists of flushing and sweating on eating in the area of skin supplied by the auriculo temporal nerve It is met with in association with parotid gland suppuration or after an operation on or a wound of the gland The nature of the reflex has not been adequately explained

## The VII Nerve

### *(The Facial Nerve)*

**Anatomy** The upper neurone has its cells of origin in the cerebral cortex in front of the Rolandic fissure in the lower third of the

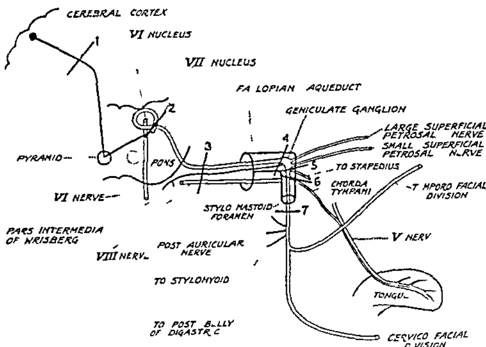



FIG 40 DIAGRAM OF THE VII NERVE

*Lesion at* 1 —*Upper Motor Neurone* Paralysis of orbicularis oris Often hemiplegia on the same side No muscular atrophy and no R D

*Lesion at* 2 —*Nuclear* Orbicularis palpebrarum and orbicularis oris paralysed Muscular atrophy and R D Hemiplegia may occur on the opposite side VI nerve often paralysed

*Lesion at* 3 —Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris VIII nerve also affected Taste affected if nerve of Wrisberg is involved

*Lesion at* 4 —Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris Auricular herpes Taste may be lost on anterior two thirds of tongue

*Lesion at* 5 —Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris Hyperacusis and loss of taste in anterior two thirds of tongue

*Lesion at* 6 —Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris Loss of taste in anterior two thirds of tongue

*Lesion at* 7 —Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris No loss of taste

precentral gyrus The fibres pass through the internal capsule near the genu and run down the mid brain in the pyramidal tract to the lower part of the pons When they reach the level of the nucleus of origin of the VII nerve they cross the mid line and terminate around the cells of origin of the VII nerve A few fibres probably end around the homolateral nucleus The VII nerve is partly motor and partly sensory The motor nucleus is situated in the lower part of the pons close to the VI nucleus The VII nerve after coursing around the VI nucleus passes out from the antero lateral aspect of the lower part of the pons in the cerebello pontine angle and runs close to the VIII nerve to enter the internal auditory meatus and the Fallopian aqueduct of the temporal bone The geniculate ganglion is situated on the VII nerve at this part of its course and shortly after the nerve to the stapedius is given off The chorda tympani nerve conveying taste sensations from the anterior two thirds of the tongue joins the VII nerve while it is still in the bone The VII nerve emerges from the skull through *the stylo mastoid foramen* *In the neck it gives off the posterior* auricular nerve to the intrinsic muscles of the pinna and to the occipital part of the occipito frontalis muscle and nerves to the stylo hyoid muscle and to the posterior belly of the digastric muscle The VII nerve then divides into the temporo facial and cervico facial branches The former supplies the corrugator supercilii the frontalis the orbicularis palpebrarum and the zygomatic muscles and the latter the buccinator orbicularis oris and platysma muscles (see Fig 40) The sensory part of the nerve is considered to be formed by the pars intermedia of Wrisberg which runs from the geniculate ganglion to the tractus solitarius portion of the glossopharyngeal nucleus in the medulla The taste fibres in the chorda tympani nerve pass from the geniculate ganglion to the nerve of Wrisberg The geniculate ganglion is also joined by the large the small and the external superficial petrosal nerves which carry sympathetic and taste fibres Further sensory fibres conveying deep pain and pressure sensations run in the peripheral divisions of the facial nerve and pass through the geniculate ganglion to the pars intermedia of Wrisberg

**Lesions of the VII Nerve** 1 *Supranuclear* The lesion may be situated in the cerebral cortex (lower part of the precentral gyrus) the corona radiata genu of the internal capsule or in the suprapontine region The lesions include a cerebral tumour abscess or vascular lesion such as a hæmorrhage thrombosis or embolus 2 *Nuclear* In the pons A tumour such as a glioma a vascular lesion encephalitis lethargica disseminated sclerosis bulbar paralysis polioencephalitis and tabes dorsalis 3 *Infranuclear* (a) *At the base of the brain* Tumours of the cerebello pontine angle basal meningitis a gumma aneurysm of the basilar artery or a fractured base (b) *In the temporal bone* Caries associated with otitis media toxæmic neuritis an operation on the mastoid (c) *In the stylo mastoid foramen* Fibrositis of the process of the posterior part of the parotid sheath which enters the foramen associated with exposure to cold (d) *In the face* A tumour or inflammation of the parotid gland as in uveo parotid tuberculosis

Injury from a wound or from forceps at birth Neuritis due to such causes as alcohol diphtheria, diabetes mellitus or leprosy

**Clinical Findings** 1 *Supranuclear Lesions* (a) *Irritative* With cortical lesions irritating the motor centre unilateral facial contractions may occur on the opposite side of the face There are often associated movements of the eyes and tongue In dementia paralytica peri oral fibrillary tremors may be noted (b) *Paralytic* There is paralysis chiefly affecting the lower part of the face of the opposite side, the upper part is much less likely to be paralysed owing to the bilateral *representation of the cortical centres in respect to their supply to the* upper part of the face Thus, although the mouth appears drawn over to the sound side and the orbicularis oris muscle is paralysed on the affected side, the patient can close both eyes and wrinkle his forehead The paralysed muscles do not atrophy and there is no reaction of degeneration Taste is not affected In addition there is frequently hemiplegia the arm body and leg being paralysed on the same side as the face when the lesion is a capsular one Although the patient cannot contract the facial muscles voluntarily on one side, he may do so under the influence of emotion Thus a joke may cause him to smile with both sides of his face, in fact the facial contraction may be exaggerated on the paralysed side This is not the case in infranuclear lesions 2 *Nuclear Lesions* There is paralysis of the upper and lower half of *the face on the same side as the lesion The muscles atrophy and* show the reaction of degeneration Further there is often paralysis of the external rectus muscle of the eye on the same side due to involvement of the contiguous VI nucleus If the pyramidal tract is also affected there is spastic hemiplegia of the opposite side of the body

3 *Infranuclear Lesions* (a) *Irritative* Severe facial spasms may occur functional tremors of the eyelids and facial tics are also of this nature (b) *Paralytic* (Bell s palsies) The clinical results vary according to the site at which the lesion is situated as described below (a) *Between the pons and geniculate ganglion* There is paralysis on the same side of the upper *and lower half of the face of the lower motor* neurone type (see below section (d)) The VIII nerve may also be involved with deafness or tinnitus or the V nerve with paralysis or sensory disturbances and if the nerve to the stapedius is affected there may be hyperacusis to sounds of low or high pitch Taste is not affected unless the nerve of Wrisberg (pars intermedia) is also involved (b) *At the geniculate ganglion* There is paralysis on the same side of the upper and lower half of the face and herpes located to the ear and external auditory meatus (herpes oticus or Ramsay Hunt syndrome) with pain may result from inflammatory lesions The chorda tympani nerve *fibres may also be affected* with *loss of taste* on the anterior two thirds of the tongue on the same side (c) *Between the geniculate ganglion and the chorda tympani nerve* There is paralysis on the same side of the upper and lower half of the face (see below section (d)) There is loss of taste on the anterior two thirds of the tongue on the same side If the lesion is proximal to the junction of the nerve to the stapedius there may be tinnitus and hyperacusis for notes of high

or low pitch Salivary secretion may be diminished on the same side
(d) *Peripheral to the junction of the chorda tympani nerve* (The usual type of Bell s palsy ) This may follow exposure to cold or sitting in a draught near an open window Both sexes are equally affected The patient is usually an adult over the age of 20 He may notice some pain or tenderness behind the ear for a day or so and then suddenly finds that he cannot move one side of the face normally and this side of the face may feel stiff He may also find that when he eats he tends to bite his cheek or lower lip on one side and the food may collect between the cheek and teeth on this side Tears may also run down the cheek on the affected side due to paralysis of the orbicularis palpebrarum muscle

*On Examination* The paralysed side of the face appears flat and expressionless the naso labial fold is less apparent The lower lid may be everted in one eye and the corner of the mouth droops Some swelling or tenderness may be found in the parotid region There is paralysis of the muscles of the upper and lower parts of the face These are tested as follows The patient is asked to shut his eyes he cannot close the eye on one side (lagophthalmos) and on trying to do so the eyeball moves upwards and either slightly inwards or outwards owing to a connection between the VII and a portion of the III nucleus If the patient is observed during sleep the affected eye may be nearly closed He is asked to frown and to raise his eyebrows and is unable to do so on the paralysed side The lower part of the face is tested by asking the patient to show his teeth the angle of the mouth is drawn outwards on the sound side and does not move out on the paralysed side He is then asked to blow out his cheeks to smile and to whistle He cannot do so on the paralysed side Spontaneous emotions also fail to provoke a contraction of the oral muscles on the paralysed side in contrast to that which may occur in organic supranuclear lesions with hemiplegia and facial paralysis Paralysis of the platysma muscle may some times be detected by making the patient depress his chin against the resistance of the examiner s hand when it may be seen by looking at the skin under the chin that the platysma does not contract on one side The affected muscles atrophy and may give a reaction of degenera tion In the course of a few days the lesion may spread up to involve the chorda tympani nerve Taste is then lost on the affected side of the anterior two thirds of the tongue It is tested for as follows The patient is given a card on which the words Sweet Salt Sour Bitter ' are written and he is told to point to the word representing the sensation he experiences when the test substance is applied to the tongue He must not withdraw his tongue into his mouth until he has indicated the sensation Sugar salt citric acid and quinine are employed as the test substances A small portion is applied in turn to the outer side of the anterior part of the tongue and gently rubbed in with cotton wool If the inflammation spreads still further up the VII nerve above the level of the junction of the nerve to the stapedius there may be tinnitus and hyperacusis to notes of high or low pitch.

Differential Diagnosis There is usually no difficulty in diagnosing

a Bell's palsy the distinctions between upper and lower motor neurone lesions described above, should be remembered In facio scapulo *humeral myopathy*, usually both sides of the face are affected and also other muscles of the body, which differentiates it from a double facial paralysis In facial hemiatrophy there is wasting but not paralysis of *the muscles of one side of the face*

**Course and Complications** The paralysis may disappear in a week or so or persist for nearly 2 years Recovery usually is first apparent in the upper half of the face *Subsequent twitching or contractures* of the affected muscles may occur when the recovery is not complete Thus the eye on the affected side may be narrow or the corner of the mouth drawn outwards "Crocodile tears" may also be expected in cases of incomplete recovery When the patient masticates and salivates, tears flow from the eye on the affected side as the result of stimulation of the lachrymal gland

**Prognosis** The prognosis in Bell's palsy depends upon the cause In the usual type due to fibrositis or periostitis there is recovery in about 80% of cases The electrical reactions are of help in giving a prognosis If the faradic and galvanic responses of the paralysed muscles are normal after 2 weeks recovery will be rapid a matter of a few weeks If there is no response to faradism and the galvanic response shows a reaction of degeneration recovery will not occur for several months If no response is obtained to faradism or to galvanism there is no likelihood of recovery If the paralysis is due to other causes such as a tumour, gumma or meningitis, the prognosis must vary with the nature of the lesion and the possibility of removing it by appropriate treatment

**Treatment** In the inflammatory type of case the patient should be kept in a warm room during the initial stages until all pain and tenderness have gone Heat should be applied locally over the stylo *mastoid foramen as by cataplasma kaolini or by fomentations* The bowels should be opened daily and a mixture given containing Pot iod gr 3 sod salicyl gr 10 sod bicarb gr 20 syr aurant m 20 aq ad fl oz 1 Fl oz 1 t d s p c Cortisone has been recommended without definite evidence of its value Strips of strapping should be placed on the affected side of the face one above and the other below the mouth to prevent the mouth being drawn over to the sound side After a week massage should be given daily to the affected muscles and later the muscles may be stimulated with a weak faradic current In all cases the Wassermann reaction should be determined and if positive a course of anti syphilitic treatment should be given Some surgeons advise early operation either by decompression of the descending portion of the nerve or by placing in the Fallopian canal a prepared nerve graft, obtained from *a cutaneous nerve of the thigh* (Ballance Duel nerve graft) The insertion of tantalum wire or ribbon in the temporal fascia to give some support to the sagging eyelids and mouth has caused improvement in some cases in which it has been tried Homo lateral stellate ganglion procaine hydrochloride block has also been reported on favourably

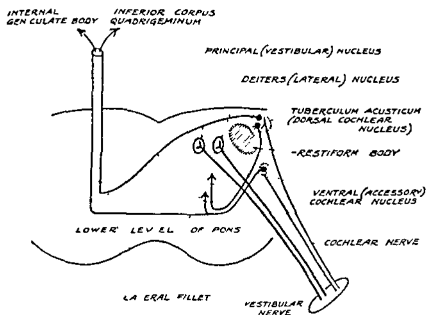



Fig 11 Diagram of the VIII Nerve Nuclei

**Bilateral Facial Paralysis** This may be congenital and there is often paralysis of the muscles supplied by the oculo motor nerves as well Other causes include double otitis media basal gummatous meningitis an aneurysm of the basilar artery diphtheria leprosy and alcoholic neuritis Both sides of the face are then expressionless

## The VIII Nerve

### (*The Auditory Nerve*)

**Anatomy** The VIII nerve consists of a cochlear and a vestibular division The former is concerned with hearing and the latter with equilibrium The cochlear nerve fibres arise from cells in the spiral ganglion situated in the central pillar of the cochlea their peripheral terminations ending in connection with the hair cells of the organ of Corti Centrally the cochlear nerve passes to the brain through the internal auditory meatus running below the VII nerve It enters the lower border of the pons on the lateral side of the restiform body and terminates around cells of the dorsal nucleus or tuberculum acusticum and the ventral or accessory cochlear nucleus A fresh relay of fibres conducts the impulses across the mid line in two strands a dorsal or striæ acusticæ and a ventral or corpus trapezoides These unite to form the lateral fillet Some of the fibres from the cochlear nuclei also run to the lateral fillet of the same side The lateral fillet ascends the mid brain and terminates in the lower auditory centres the internal geniculate body and the inferior corpus quadrigeminum The internal geniculate bodies are connected by Gudden s commissure A further relay conveys the impulses through the posterior part of the internal capsule to the higher auditory centres in the superior temporal gyrus of the cerebral cortex

The cells of origin of the vestibular nerve are in the ganglion of Scarpa which lies in the internal auditory meatus Their peripheral terminations are in the semicircular canals and the otolith organs (the utricle and saccule) The fibres from the semicircular canals convey sensations of movement (kinetic impulses) whereas those from the otolith organs transmit sensations of position (static impulses) Centrally the fibres pass with the cochlear division and enter the lower part of the pons between the restiform and olivary bodies terminating around the cells of the principal or vestibular and Deiters or the lateral nucleus The upper part of Deiters nucleus constitutes Bechterew s nucleus (see Fig 41) The further course of the vestibular path is rather open to discussion Fibres pass from Deiters nucleus up the pons and mid brain in the medial longitudinal bundle In this way connection is made with the nuclei of the oculo motor nerves thus establishing relationship between equilibrium and eye movements Impulses are also carried downwards in the medial longitudinal bundle to connect with the XI nucleus and establish a relationship with equilibrium and head movements Fibres also descend the cord in the vestibulo spinal tract whereby a connection is made between the sense of equilibrium and the tone of skeletal muscles Some fibres of the

vestibular nerve run direct to the cerebellum by the inferior cerebellar peduncle and fibres also pass from the cerebellum by *Edinger's tract* in the inferior cerebellar peduncle to Deiters' nucleus (see Fig 31)

**Lesions of the VIII Nerve** The VIII nerve may be affected by tumours of the cerebello pontine angle pressing on or growing from the nerve itself, or by basal meningitis due to syphilis or cerebro spinal fever Neuritis may be due to toxins of typhoid or scarlet fever to syphilis or to such substances as alcohol tobacco quinine or salicylates Atrophy may occur in tabes dorsalis or in disseminated sclerosis The nerve may be injured in a fracture of the base of the skull or by disease of the petrous bone Inflammation may affect it in otitis media labyrinthitis or Eustachian catarrh It may be involved in a vascular lesion such as a hæmorrhage or thrombosis and by a leukæmic infiltration Central lesions are rare Deafness sometimes results from streptomycin injections A bilateral lesion of the superior temporal gyrus may cause deafness or word-deafness may result from an unilateral lesion affecting the left superior temporal convolution In hysteria there may be complete deafness

**Clinical Findings** Lesions of the VIII nerve may result in deafness tinnitus or vertigo *Deafness* may be perceptive or conductive In *perceptive deafness* there is interference with the perceptive mechanism in the cochlea or with the transmission of the impulses to the cortex This may be due to a lesion of the inner ear of the VIII nerve or of the brain The characteristic findings in this type of deafness are loss of high tone, as shown by the audiogram a positive Rinne test (aerial conduction is better than bone conduction) subnormal absolute bone conduction a negative Weber test (the tuning fork placed on the vertex is lateralised to the sound or better ear) and a lowering of the upper tone limit

In *conductive deafness* there is interference with the passage of sound waves to the inner ear the lesion being in the external meatus or middle ear The characteristic findings are a low tone loss a negative Rinne test (bone conduction is better than aerial) a positive Weber test (the tuning fork placed on the vertex is lateralised to the affected or the worse ear if both are involved) and the absolute bone conduction is normal

The following tests should be carried out when examining a patient who complains of deafness 1 The external ear is examined for wax or other foreign bodies 2 The tympanic membrane is inspected for evidence of disease of the middle ear 3 Hearing is tested by whispered and conversation voice Alternate ears are tested with one ear closed tightly the distance being recorded in feet or inches 4 Rinne test A 512 cycles per second tuning fork is placed vibrating on the mastoid process and the meatus closed with a finger When the fork is no longer heard it is held close to the meatus Normally vibration can be heard again for a short period This means that air conduction is greater than bone conduction (Rinne positive) 5 Weber test The vibrating tuning fork is placed on the vertex of the head, and the patient is asked in which ear he hears the vibrations Normally they are heard

equally in both ears and the patient hears them in the centre of the head Further if one ear is closed the vibrations appear louder on that side 6 Absolute bone conduction The vibrating tuning fork is placed on the mastoid process with the ear closed by the finger When the patient no longer hears the fork it is placed on the examiner s mastoid with the ear closed If the examiner hears it the time is noted during which the sound persists 7 Lower tone limit Loss is shown by failure to hear a vibrating fork of 24 cycles per second 8 High tone limit A normal person should hear up to 16 000 cycles per second Any marked lowering indicates a pathological process in the inner ear It is best tested with a monochord 9 The audiogram The audiometer is an instrument in which the pure tones are produced electrically and these can be varied in pitch and intensity by dials on the instrument To test air conduction the ear phones are applied to both ears and the ear not to be tested is cut out from the electrical circuit Bone con duction is also tested a single plastic receiver being applied over the mastoid process first on one ear and then on the other The patient is tested against the different frequencies and presses a button which lights a lamp directly he hears a sound Eight tones are produced with frequencies of 64 to 8 192 cycles per second in octave steps These are shown along the top of the audiogram and correspond with the musical notations of C c $c^1$ $c^2$ $c^3$ $c^4$ $c^5$ $c^6$ 256 being the middle C of the piano The intensity of each tone is increased until heard by the patient and then charted on an audiogram In perceptive deafness there is loss of high tone in conductive deafness loss of low tone The frequencies 256 512 and 1 024 are those of the ordinary speaking voice The average of these three readings multiplied by 0 8 gives the percentage hearing loss for the spoken voice *Paracusis* means that the patient hears best in a noise It is found especially in otosclerosis

*Tinnitus aurium* The chief symptom complained of may be noises in the ear or head with or without some degree of deafness This may be due to general causes such as anæmia a high or low blood pressure or neurasthenia or to local affections especially to otosclerosis chronic otitis media bony overgrowth of the external meatus wax in the ears labyrinthine lesions or intracranial suppuration It may occur as an aura in epilepsy or be produced by drugs such as quinine and salicylates The noises may be continuous or intermittent and of varying character often hissing or roaring or at times higher pitched or pulsating in correspondence with the heart beat In all cases a complete local examination should be made by an otologist as well as a general examination of the patient by a physician

**Treatment** This consists in local measures for relief of Eustachian catarrh etc which falls in the province of the specialist and in addition bromides in doses of gr 10 to 20 may be given 3 times a day

**The Vestibular Nerve** Lesions of the vestibular nerve or of its terminations in the semicircular canals and otolith organs result in three main symptoms Vertigo postural or kinetic deviation and nystagmus

**Vertigo** The patient who complains of vertigo may say that

his own body appears to be moving or that surrounding objects are moving Vertigo may thus be called, perhaps incorrectly, subjective or objective

**Etiology** The common causes of vertigo include Wax in the ears otitis media, labyrinthitis, alimentary disturbances, train car or sea sickness, migraine, cerebellar lesions, high blood pressure over smoking alcohol and epilepsy Vertigo may also be psychogenic associated with an anxiety state, ocular as when looking down from a height or due to external oculo motor paralysis It is sometimes met with in brain stem lesions such as disseminated sclerosis a tumour or a vascular disturbance A cysticercus in the IV ventricle may also produce attacks of severe vertigo Vestibular vertigo results from irritative lesions of the ear affecting the semicircular canals Epidemic vertigo, with uncontrollable vomiting is also described This may be a manifestation of influenza Streptomycin may cause giddiness from involvement of the vestibular nerve or its nucleus Relief may be obtained by ice cold water bottles applied to the occipital region, and by the administration of dextrose 1 oz t i d by mouth

**Postural Deviation** The patient may tend to fall towards the affected side in irritative vestibular lesions, when he stands with eyes closed and feet together

**Kinetic Deviation** This is described later (see below)

**Nystagmus** Irritative labyrinthine lesions produce spontaneous vestibular nystagmus to the opposite side, *i e*, the eyes make a slow movement to the same side and a rapid twitch back to the opposite side These movements are increased when the patient looks to the side of the nystagmus (*i e*, that in which the rapid movement occurs)

**Clinical Findings** Certain tests are applied in suspected cases of labyrinthine lesions These include 1 *The Rotation Test* The patient is placed in a special chair with a head rest so that the head is inclined forwards 30° to the vertical plane He is then rotated 10 times in 20 seconds and the chair stopped Rotation is done in both directions with a suitable interval between On rotating to the right and stopping normally the slow component of the nystagmus the tendency to fall and the past pointing (Bárany's test) are to the left, the quick component of the nystagmus and the sensation of vertigo are to the right With rotation to the left the opposite results are found Diminished responses on rotation to one side indicate a lesion of the opposite labyrinth

2 *The Caloric Test* The patient lies on a couch with the head tilted back 60° from the vertical plane The drum is first inspected to see that it is not perforated and that the external auditory meatus is free from wax and polypi A douche can holding at least one pint of water, is slung so that the bottom of the can is 2 feet above the head of the patient The water is delivered through a rubber tube connected with an ear nozzle with a bore of 4 mm Each ear is first tested with cold water at 7° below normal *i e* 30 C and the water is delivered into the external meatus for 40 seconds, using a stop watch The patient keeps his eyes fixed on a spot high up on the opposite wall A normal response

shows a quick nystagmus to the opposite side The ears are next tested with hot water at 7° above normal *i e* 44 C The normal response is the quick component of the nystagmus to the same side An absent or diminished response on one side indicates a lesion of that labyrinth Cold air may also be used as a stimulus the air being blown through a cooled metal tube (Dundas Grant tube) cooled by a spray of ethyl chloride

**Irritative Vestibular Lesions** These are exemplified in Ménière s syndrome and in Ménière s symptom complex. *Ménière s syndrome* The disease originally described by Ménière was due to an acute labyrinthine hæmorrhage associated with acute purulent labyrinthitis and was not typical of what we now call Ménière s disease In some cases there is pressure on the auditory nerve caused by an aneurysm of the basilar artery an abnormally large internal auditory artery or by a cerebello-pontine angle tumour It is probable that only lesions affecting the vestibular division of the VIII nerve cause Ménière s disease Some authorities believe that it may be caused by faulty water or salt metabolism resulting in a water logged labyrinth or that the hydrops of the endolymphatic labyrinth is in some cases an allergic manifestation The patient who is usually an adult is suddenly attacked with severe vertigo and falls There may be a preliminary phase of low pitched tinnitus Vomiting may occur

*On Examination* The patient is usually found lying on the sound side if he attempts to turn on his back or on the affected side vomiting is induced The eyes show coarse nystagmoid movements to the affected side There may be momentary loss of consciousness Permanent deafness usually ensues *Ménière s symptom complex* This may be due to vascular spasm of the internal auditory artery causing alteration in the tension of the endolymph or to otosclerosis The attacks resemble those of the Ménière s syndrome but between the attacks there is no loss of hearing

**Treatment** During an acute attack an injection of hyoscin hydro bromid gr 1/100 should be given Between attacks various drugs may be tried Quinine sulphate in doses of gr ½ t d s or phenobarbitone gr ½ t d s may be administered to diminish the sensibility of the labyrinth In some cases relief is obtained by the administration of a salt poor diet (see p 474) with restriction of the fluid intake to 40 oz in the 24 hours Ammon chlorid 0 5 G tablets 6 t i d with meals should be given for 3 days out of every five Mersalyl 2 ml may also be injected intramuscularly once a week Favourable results have also been reported by the administration of Pot chlorid gr 15 aquam ad m. 60 m 60 in water 6 to 8 times a day The dietary should then have a normal sodium content Avomine tab mg 25 t i d may be of value in some cases and in others nicotinic acid tab mg 50 1 to 3 t i d is helpful Surgical treatment consists in division of the auditory nerve or of the vestibular branch alone or labyrinthotomy Determination of the side affected is not always easy The tinnitus and deafness are usually more severe on one side and the caloric tests may indicate which labyrinth is at fault Vertigo is not always and tinnitus is rarely

relieved by the operation Surgery should only be advised if medical treatment fails

**Acute Vestibulitis** This is due to acute inflammation of the cochlea and labyrinth There is tinnitus, vertigo vomiting and usually pyrexia, and it often results in permanent deafness

## The IX Nerve

*(The Glosso pharyngeal Nerve)*

**Anatomy** The IX nerve is both motor and sensory in function The motor fibres arise from the nucleus ambiguus in the medulla This nucleus is common to the IX and X nerves The nucleus ambiguus extends through the medulla from the level of the VIII nerve above to the decussation of the fillet below The sensory fibres arise from cells in the superior (jugular) and petrosal ganglia These ganglia lie on the nerve trunk in the jugular foramen The sensory fibres pass into the medulla and run down it as the tractus solitarius the fibres ending at different levels in the nucleus of the tractus solitarius which adjoins the tract (see Fig 42) Other ascending fibres enter the dorsal nucleus and pass up the medulla The further central course of the sensory fibres is not definitely known It will be remembered that the fibres of the pars intermedia of Wrisberg also end in the tractus solitarius (see p 391) The sensory and motor fibres of the IX nerve enter and leave the medulla in 5 or 6 strands on the postero lateral surface between the olivary and restiform bodies The nerve passes through the jugular foramen with the X nerve, but in a special sheath of dura mater It runs between the internal and external carotid arteries and then deep to the hyoglossus muscle to the pharynx

*Sensory Branches* In the skull it receives a tympanic branch (Jacobson s nerve) from the petrosal ganglion, which supplies the middle ear the mastoid air cells and the Eustachian tube It conveys taste sensation from the posterior third of the tongue and from taste buds on the soft palate, epiglottis and arytenoid cartilages It transmits common sensation from the back of the tongue tonsil, part of the soft palate and the upper part of the pharynx

*Motor Branches* The IX nerve supplies motor fibres to the stylo pharyngeus palatopharyngeus and palatoglossus muscles and secretory fibres to the parotid gland The constrictors of the pharynx are probably supplied by the vagus

**Lesions of the IX Nerve** Nuclear lesions may be due to such causes as a tumour a gumma, syringomyelia disseminated sclerosis polioencephalitis or thrombosis of the posterior inferior cerebellar artery There is no definite pathology of central lesions

**Clinical Findings** *Motor Lesions* It is probable that lower motor neurone lesions causing dysphagia are due to affections of the X nerve A lesion paralysing the stylopharyngeus muscle may cause some dysphagia, as the larynx is not then drawn up on swallowing and food may enter the trachea

*Sensory Lesions* Loss of taste over the posterior third of the

tongue with anæsthesia of the pharynx and tonsillar region and loss of the pharyngeal reflex may result from involvement of the tractus solitarius in the medulla

*Glossopharyngeal Neuralgia* The patient is usually an adult over middle age who complains of pain often agonising on swallowing or talking in the region of the pillars of the fauces on one side The pain may shoot to the ear and disturb sleep Pressure on the tonsil on the affected side may produce a paroxysm of pain If the tympanic branch alone is affected, there is deep seated pain in one ear which occurs in paroxysms but is not aroused by swallowing

Treatment There is usually no treatment available for nuclear lesions unless due to syphilis Glossopharyngeal neuralgia should be treated first with a gelsemium mixture, as for trigeminal neuralgia The inhalation while the patient lies down of trichlorethylene m 15 t d s may afford relief This is put up as a sterule encased in cotton wool and silk It can be broken between the fingers and the vapour *inhaled* *If this fails an operation should be considered* *The glosso* pharyngeal nerve may be divided intracranially or it may be exposed at its emergence from the jugular foramen and avulsed from its central connection with the medulla

## The X Nerve

### (*The Vagus* *The Pneumogastric Nerve*)

Anatomy The vagus nerve contains motor and sensory fibres The bulbar portion of the spinal accessory nerve is considered to be a part of the vagus nerve The motor fibres arise from the nucleus ambiguus in the medulla (see Fig 42) The sensory fibres are derived from cells in the trunk ganglion (ganglion nodosum) which lies on the nerve just external to the skull The sensory fibres enter the medulla and terminate in the tractus solitarius (descending) and in the dorsal nucleus (ascending) (see Fig 42) The upper motor neurones arise in the cerebral cortex in the lower part of the precentral gyrus and pass down in the pyramidal tract to end around the motor nuclei of the opposite side The vagus nerve emerges from the side of the medulla by a series of roots in line with those of the IX nerve above A meningeal and an auricular branch leave the nerve at the level of the root ganglion The latter Arnold s nerve supplies the skin at the back of the pinna and the external auditory meatus The vagus leaves the skull through the jugular foramen behind the IX nerve Two branches arise from the trunk ganglion the pharyngeal and the superior laryngeal nerves The bulbar fibres of the XI nerve join the X nerve just below the trunk ganglion The vagus runs down the neck in the carotid sheath and gives off the recurrent laryngeal branch and cardiac branches It enters the thorax passing between the internal carotid and subclavian arteries on the left side and crossing in front of the subclavian artery on the right side It then traverses the superior and posterior mediastinal spaces In the thorax it gives rise to the pulmonary and œsophageal plexuses The vagus enters the abdomen through the

œsophageal opening of the diaphragm, the left nerve being in front of and the right nerve behind the œsophagus It terminates in branches to the stomach and small intestine Motor fibres are supplied to the soft palate, the pharynx larynx œsophagus, stomach small intestine trachea and bronchi The muscles of the palate, pharynx and larynx are probably supplied by the bulbar portion of the XI nerve The nerve supply of the laryngeal muscles is described on p 131

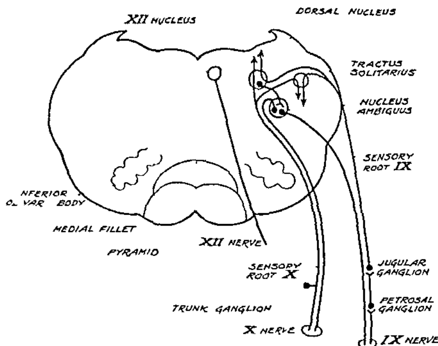



FIG 12 DIAGRAM OF THE IX X AND XII CRANIAL NERVE NUCLEI

Secretory fibres are conveyed to the stomach small intestine and pancreas Inhibitory fibres are carried to the heart Sensory impulses pass by the vagus from the skin of the pinna and external auditory meatus and from the mucous membranes of the larynx respiratory tract, œsophagus and stomach

**Lesions of the X Nerve** *Nuclear Lesions* may be due to progressive bulbar palsy, syringomyelia encephalitis lethargica rabies thrombosis disseminated sclerosis, or syphilis *The nerve* may be injured at various sites such as (*a*) *In the skull* This may be due to basal meningitis especially syphilitic, a tumour, an aneurysm of the vertebral artery, or a fractured base (*b*) *In the neck* The lesion may be due to a wound, a tumour or to damage at an operation (*c*) *In the thorax* An aneurysm a mediastinal tumour or enlarged glands may compress the nerve

The vagus nerve may be affected by neuritis, especially in diphtheria Less often the neuritis is due to typhoid fever influenza, pneumonia, or to chemicals such as alcohol or arsenic

**Clinical Findings** (*a*) *Irritative Lesions* Irritation of the vagus in the mediastinum by enlarged glands or a tumour may cause an intractable cough somewhat resembling whooping cough Attacks of laryngeal spasm, as in the laryngeal crises of tabes may be due to central lesions Reflex laryngeal spasm also occurs in such conditions as laryngismus stridulus Irritation of the vagus may also cause hyperæsthesia of the posterior wall of the external auditory meatus Touching the skin in this area may provoke cough (the tragus sign) Some instances of dyspnœa on slight exertion or of bradycardia may be due to vagal stimulation and asthma is thought to be associated with undue irritability of the broncho motor portion of the vagus nucleus Irritation of the pharyngeal fibres of the vagus may account for the dysphagia in hydrophobia or in hysteria Similarly the gastric crises of tabes may be due to irritation of the gastric branches of the vagus

(*b*) *Paralytic Lesions* Paralysis of the vagus nerve on one side results in anæsthesia and paralysis of the palate and larynx on the same side (the syndrome of Avellis) The palatal paralysis is detected by inspecting the palate when the patient says ah The raphe of the palate and the uvula then deviate to the sound side The oculo cardiac reflex is also usually abolished on that side This is tested by pressing on the eyeball with the eyelid closed Normally the pulse rate should slow by about 10 to 20 beats a minute With a vagal paralysis this reflex slowing may be absent With bilateral lesions affecting the palate there is usually nasal regurgitation and a nasal voice With pharyngeal paralysis there is dysphagia and liquids often enter the larynx and provoke cough The effects of paralysis of the laryngeal branches of the vagus are described on p 132

## The XI Nerve

### (*The Spinal Accessory Nerve*)

**Anatomy** The bulbar portion of this nerve has been described above under the X nerve of which it constitutes a part The spinal portion is motor in function and arises from a column of cells in the lateral part of the anterior horn of grey matter of the spinal cord from the first to the fifth cervical segments The nerve fibres emerge between the anterior and posterior nerve roots and joining to form the XI nerve ascend by the side of the cord in the subdural space to the foramen magnum It is here joined by the bulbar portion of the nerve which again shortly leaves it to pass to the vagus nerve just below the trunk ganglion The XI nerve leaves the skull through the jugular foramen with the X nerve and passes between the internal carotid artery and the internal jugular vein It then runs backwards through the sternomastoid muscle leaving its posterior border at the level of the junction of its upper and middle thirds It finally crosses the posterior triangle of the neck to the under surface of the trapezius muscle The XI nerve supplies the sternomastoid and part of the trapezius muscles The trapezius is supplied in its upper part by the XI nerve

and to a lesser degree in its lower part, the central portion being innervated by the sub trapezial plexus from the 3rd and 4th cervical nerves

**Lesions of the XI Nerve** 1 *Nuclear*, as in progressive muscular atrophy, disseminated sclerosis, syringomyelia or myelitis 2 *At the base of the brain*, the lesion being due to meningitis aneurysm of the vertebral artery, or a fractured base 3 *In the neck* Trauma by lifting heavy weights or by a wound Injury during an operation for glands Cervical caries Neuritis is not a common cause

**Clinical Findings** Irritative lesions of the XI nerve or of its supranuclear connections may cause spasmodic torticollis Paralytic lesions The sternomastoid or trapezius muscles may be paralysed individually or together With unilateral sternomastoid paralysis the patient does not notice any weakness

*On Examination* No deformity is visible, but if the patient is asked to turn his head away from the affected side while the examiner s hand is held against his chin on the sound side, the sternomastoid muscle does not contract and stand out on the affected side If both sternomastoids are paralysed the head is inclined to fall backwards When the trapezius on one side is paralysed the patient notices difficulty in raising the corresponding arm above the horizontal in shrugging the shoulder or in causing the scapula to approach the mid line There is drooping of the shoulder on the affected side The scapula is in an abnormal position being farther from the mid line and lower than normal and rotated outwards The vertebral border is prominent and runs from below upwards and outwards, this is known as winging' of the scapula and is best seen when the arm is held forwards and below the horizontal level When the patient tries to shrug his shoulders or to approximate the scapulæ, the trapezius muscle can be seen and felt to remain uncontracted on one side If both trapezii are paralysed the head is inclined to fall forwards Fibrillary twitchings may be seen in the paralysed muscles which also give a reaction of degeneration

## The XII Nerve

### (*The Hypoglossal Nerve*)

**Anatomy** The nucleus of origin is situated in the medulla near the floor of the IV ventricle, extending from the level of the striæ medullares above to the pyramidal decussation below (see Fig 4?) The nerve fibres leave the medulla between the pyramid and the olive in a series which unite to form three roots These join and the hypo glossal nerve leaves the skull through the hypoglossal canal (anterior condylar foramen) The nerve passes between the internal carotid artery and the internal jugular vein, and forming connections with the first and second cervical nerves, reaches the floor of the mouth to supply the intrinsic and extrinsic muscles of the tongue

**Lesions of the XII Nerve** The lesions may be 1 *Supranuclear* These are usually vascular in the cerebral cortex or internal capsule 2 *Nuclear* Various lesions may be found such as those due to pro

gressive bulbar paralysis syringobulbia polioencephalitis disseminated sclerosis thrombosis a gumma or a tumour 3 *At the base of the brain* The nerve roots may be affected by a tumour by meningitis an aneurysm of the vertebral artery or a fractured base 4 *In the hypoglossal canal* Periostitis may cause compression of the nerve 5 *In the neck* Enlarged glands or caries of the first cervical vertebra Neuritis rarely occurs in association with alcohol or lead

**Clinical Findings** 1 *Upper Motor Neurone Lesions* There is weakness and spasticity of the affected half of the tongue On protrusion it deviates towards the paralysed side There is usually hemiplegia of the same side of the body

2 *Lower Motor Neurone Lesions* Nuclear lesions are often bilateral as in progressive bulbar palsy There is difficulty in chewing and swallowing and in speech The lips and palate are often affected as well The lesion may involve one or other pyramidal tract with hemiplegia The tongue lies flaccid and shrivelled on the floor of the mouth

*With Unilateral Lesions* The affected half of the tongue is small and shrivelled On protrusion the tongue deviates to the weakened side Fibrillary contractions may be seen on the affected side of the tongue and a reaction of degeneration may be present Speech is not usually affected

## SYPHILIS OF THE NERVOUS SYSTEM

**Introductory** The incidence of neuro syphilis has fallen considerably during the last 20 years or so Syphilitic lesions of the central nervous system are usually divided into two groups meningo vascular (interstitial) and parenchymatous The former includes such clinical conditions as meningitis meningo encephalitis meningo myelitis cerebral and spinal gumma cerebral syphilis and spinal syphilis and the latter group embraces general paralysis of the insane and tabes dorsalis The distinction is however somewhat artificial as the primary cerebral lesion in either case is invariably an arteritis or a lymphangitis Thus the meninges and vessels are affected predominantly in meningo vascular syphilis but in general paralysis of the insane and in tabes lesions can be demonstrated at autopsy in the vessels and meninges and during life the cerebro spinal fluid shows a meningeal reaction (increase of cells etc )

The nervous system is believed to be infected during the primary or secondary stages of syphilis in cases which subsequently develop neuro syphilis although not every case so infected shows signs later of neuro syphilis Actually there is evidence of infection of the nervous system in many cases of syphilis during the primary and secondary stages Thus there may be headache giddiness and some disturbance of vision The cerebro spinal fluid shows an increase of cells up to 100 per c mm globulin may be present and treponemes found in the fluid The Wassermann reaction is also at times positive in the fluid before it is so in the blood and a luetic curve is obtained with Lange s test (see Fig 24) In the majority of cases these changes are transitory

but the treponemes may lie dormant and after 4 or 5 years another meningeal reaction occur, with clinical evidence of neuro syphilis

### Meningo-vascular Syphilis

The lesions may affect chiefly the brain or the spinal cord

### Cerebral Syphilis

**Pathology** The meninges may be chiefly affected the pia arachnoid being involved in a gummatous infiltration The base of the brain is a favourite site, and various cranial nerves may be affected In other cases the lesion is cortical Small or large gummata form and compress the underlying brain substance The vascular changes consist of endarteritis obliterans whereby the blood supply is cut off from portions of the brain with consequent softening, hæmorrhages or the formation of cysts

**Clinical Findings** The patient may give a history of syphilitic infection 4 or 5 years previously He complains of headache which is often worse at night, the memory deteriorates and transient pareses of the limbs or double vision may occur

*In cortical meningitis* there may be epileptiform convulsions affecting one or more limbs, and during the attacks the patient may or may not lose consciousness

*In basal meningitis* various cranial nerve lesions can be detected such as optic neuritis unequal irregular or fixed pupils ptosis weakness of the external ocular muscles pains in the face, deafness weakness of one side of the tongue or unilateral vocal cord paralysis

*A cerebral gumma* gives rise to the signs of a cerebral tumour, there is optic neuritis and often vomiting with headache

*Syphilitic dementia* may occur in meningo vascular syphilis, the symptoms closely resemble those of general paralysis but there is usually evidence of some cranial nerve lesion

### Spinal Syphilis

**Pathology** The dura may be much thickened especially in the cervical region, giving rise to pachymeningitis cervicalis hypertrophica There is usually compression of the cervical nerve roots In other cases there is a gummatous infiltration of the pia arachnoid especially in the thoracic region The pia becomes adherent to the cord and the vascular supply of the cord is interfered with A condition of meningo myelitis then results Thrombosis or a hæmorrhage in the cord will give rise to acute syphilitic myelitis A gumma may also form with symptoms of a cord tumour

**Clinical Findings** *Pachymeningitis Cervicalis Hypertrophica* The patient complains of severe pains in the neck and upper part of the back and arms with weakness of the arm muscles

*On Examination* There is wasting of the arm muscles with diminution of the deep reflexes and some loss of cutaneous sensation

*Chronic Meningo myelitis* The patient complains of pain in the

back usually in the thoracic region with weakness and numbness of the legs and often loss of sphincter control

*On Examination* Signs of a lesion of the cord in the thoracic region are found Thus there is weakness of the legs or paraplegia with spasticity of the leg muscles and increase of the deep reflexes The plantar responses are extensor The abdominal reflexes are lost below the site of the lesion There is some diminution of sensation or anæsthesia over the legs and lower half of the body below the lesion The disease runs a course usually lasting a few months when with suitable treatment recovery may gradually take place

*Erb s Syphilitic Paralysis* This usually develops insidiously several years after infection there is spasticity of the legs without marked changes of sensation

*Acute Transverse Myelitis* The patient is suddenly taken ill with weakness or paralysis of the legs and loss of sphincter control The signs closely resemble those d scribed above for meningo myelitis but the paralysis is usually of a flaccid type in the early stages with loss of deep reflexes and of the plantar response Later a spastic paralysis may ensue with exaggerated tendon reflexes and extensor plantar responses A zone of hyperæsthesia is usually found running round the body at the skin level corresponding with the site of the lesion in the cord In both these varieties there is often evidence of some intracranial involvement such as headache giddiness and weakness of the external ocular muscles

**Differential Diagnosis** The effects produced by meningo vascular syphilis may and often do closely simulate many diseases of the nervous system such as epilepsy Jacksonian epilepsy a cerebral tumour meningitis due to other causes general paralysis tabo paresis dis seminated sclerosis progressive muscular atrophy etc The presence of cranial nerve palsies is always suggestive of a syphilitic infection The diagnosis is established by the results of the examination of the blood and of the cerebro spinal fluid The blood Wassermann reaction is nearly always positive and the cerebro-spinal fluid shows a positive Wassermann reaction increase of cells the presence of globulin and often a meningitic curve (see Fig 24)

**Prognosis** This is most hopeful when the lesions are meningeal rather than vascular

**Treatment** Penicillin is now the sheet anchor of treatment bismuth and malaria are seldom used The first injection should be small 1 500 units of crystalline penicillin intramuscularly t i d to avoid provoking a Jarish Herxheimer focal reaction (see p 596) Subsequently the dose is increased to 60 000 units every 3 hours for 24 hours then to 100 000 units every 3 hours for 24 hours and then to 500 000 units of crystalline penicillin or 600 000 units of procaine penicillin every 12 hours for 10 to 12 days The penicillin eradicates the treponemes from the cerebral nervous system but to deal with the inflammatory products it is customary to give an iodide and mercury mixture during the penicillin treatment and for 3 to 4 weeks after The mixture contains Pot iod gr 10 liq hydrarg perchlor m 30 liq arsenical m 2 aq chlorof

ad fl oz ¼ Fl oz ½ ex aq t d s p c A lumbar puncture should be performed after 3 months If there are more than 4 lymphocytes per c mm a second course of penicillin is given If the cell count remains below 4 lymphocytes for 6 months it is not likely to rise again In cases of primary optic atrophy with progressive loss of vision it is customary to give also a course of malaria fever treatment The patient is infected with benign tertian malaria This can be done either by the bite of an infected mosquito or by the intramuscular injection between the scapulæ, of 5 ml of citrated blood taken from a patient suffering from malaria After about 7 to 10 days if the infection takes the patient develops an attack of malaria He is allowed to suffer about 8 such attacks and quinine or Atebrine is then given to abort the course of the malaria

### Parenchymatous Syphilis

This includes tabes dorsalis and general paralysis of the insane

### Tabes Dorsalis

### (*Locomotor Ataxia*)

**Definition** *A disease characterised by pains alteration of sensation* hypotonus inco ordination loss of deep reflexes and trophic and visceral changes

**Etiology** *Tabes dorsalis is caused by infection with the Treponema* pallidum The infection is generally acquired in adult life In juvenile tabes the disease is congenital or rarely due to an accidental infection of *the child Only about 1 to 5% of those infected with syphilis develop* tabes *Predisposing causes* 1 Age Between 20 and 50 years 2 Sex Males predominate except in juvenile tabes An injury appears in some *cases to light up the disease*

**Pathology** In the majority of cases of syphilis there is infection of the central nervous system during the secondary stage The infec *tion may die out or lie dormant* and after several years the treponemes may give rise to lesions of the posterior nerve roots of the spinal cord, usually in the lumbo sacral region The lesion is essentially a fibro blastic infiltration of the sheath of the posterior nerve roots, which spreads into the nerve, and in which treponemes are demonstrable The exogenous nerve fibres whose cells of origin are situated in the posterior root ganglia are thus affected and degenerate The fibres conveying sensations of pain touch and temperature end in the substantia gelatinosa Rolandi on entering the cord and a fresh relay carries the impulses up in the spino thalamic tracts The latter tracts do not degenerate (except in advanced cases), as they are composed of endogenous fibres although their impulses are affected owing to interference with the exogenous fibres in the posterior roots Sclerosis of the posterior columns results from the overgrowth of the neuroglial tissue In cervical tabes the changes are most marked in the upper part of the cord The I and II cranial nerves may be affected by a peri vascular infiltration with lymphocytes and plasma cells *Less frequently* the V, VIII X and XII nerves are involved by a fibroblastic infiltration

Degeneration of the colliculo nuclear fibres in the mid brain connecting the higher visual centres in the superior corpora quadrigemina with the III nucleus is thought to give rise to the Argyll Robertson pupils (see Fig 33) Alternatively it has been suggested that the ciliary ganglion is affected the fibres which cause accommodation being thought to travel to the ciliary body without passing through the ciliary ganglion and so they are not involved The bones may be rarefied and the joints show atrophic or hypertrophic bony changes with fluid formation in the synovial membrane (Charcot s joints see p 410) In some cases the anterior roots of the cord show degenerative changes

Clinical Findings The patient is usually an adult male who may or may not give a history of syphilitic infection some years previously He may complain first of an abnormality in micturition such as delay in beginning or less often of frequency or incontinence Impotence may also be an early symptom In other cases pains have been noticed either prolonged muscular ones which are probably described as rheumatic or sharp agonising lightning pains occurring in attacks of sharp stabs These are often felt near the outer side of the knee in the calf heel or foot or the pains may run up and down the leg In other cases the first symptom is dimness of vision or transient diplopia or there may be complaints of difficulty in walking the foot appearing to catch in the ground or unsteadiness may be noted, especially in the dark or when the eyes are closed as in washing the face Numbness tingling or sensations of cold may be felt in the legs or trunk or a sense of constriction or pain around the trunk often at the level of the upper part of the abdomen (girdle sensation) Various tabetic crises may cause distress Very acute pain may occur in the epigastrium with nausea vomiting and faintness or even hæmatemesis (gastric crises) Intestinal crises are characterised by attacks of diarrhœa rectal crises by tenesmus vesical and renal crises by suprapubic pain and frequency urethral crises by pain in the urethra nasal crises by sneezing and laryngeal or bronchial crises by dyspnœa and cough Præcordial pain may occur with cardiac crises

*On Examination* The physical signs found in an early case are very variable and few cases pass through clearly defined pre-ataxic, ataxic and paralytic stages The ankle jerks are lost early Areas of altered cutaneous sensation may be detected such as patches of anæsthesia to the pin prick light touch or temperature especially over the tibiæ the perineum the inner side of the arms the tip of the nose or on the chest from the level of the second costal cartilage to the xiphisternum There may be definite delay in appreciating painful stimuli Hyperæsthesia to light touch may be present especially over the trunk. The bone vibration sense may be lost in the feet or legs The pupils may show the Argyll Robertson sign being small and the reaction to light being lost, while that to accommodation persists The pupils may be irregular unequal and pin point in size Ataxia may be demonstrated by asking the patient to stand with his feet and toes together and eyes shut The patient may sway or fall (Romberg s sign) and should be carefully watched during the test In a less advanced stage he is unable

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becomes smaller than does the pupil in the other eye After ion of the accommodation effort re dilatation is often slow the aking several minutes to attain its original size Markus further l that the pupil reacts to accommodation and not to convergence kle jerks knee jerks and the arm jerks are absent in many cases use is unknown

ficulty in walking may occur in disseminated sclerosis but here intar response is extensor and this also applies to Friedreich s In cerebellar ataxia there is usually nystagmus and the ataxy increased when the eyes are shut Syphilitic meningo myelitis osely simulate tabes but the course is usually more acute

**irse and Complications** The course is very variable and the may be arrested at any stage whereas an acute intercurrent may accelerate its progress In the elderly it tends to run a course Optic atrophy usually means that the patient will be n a few years Complications include cystitis and pyelonephritis ation of the hip may result from atrophy of the head of the femur al paralysis of the insane sometimes occurs as a complication

**ognosis** This is very variable Death may rapidly ensue the t wasting and becoming bedridden or developing some com on such as an intercurrent disease or infection of the urinary In other cases the disease is arrested for long periods

**atment** A preliminary period of rest in bed for a few weeks is of value A course of treatment similar to that described on for meningo vascular syphilis should be given Ataxia can be ed by having the soles of the shoes weighted and the patient can ned to regulate his movements by means of his eyes by Fraenkel s ses The patient practises placing his feet in certain positions and tain marks both when he is lying and on standing When the is arrested or burned out the cerebro spinal fluid usually an absence of globulin and no colloidal gold curve and the cell is low the Wassermann reaction is usually negative Gastric may be treated by the subcutaneous injection of 1 ml of 1/1 000 aline hydrochlor If relief is not obtained an intravenous injection l of 10% calcium gluconate or of atropin sulph gr 1/100 may successful Laryngeal crises can generally be relieved by an tion of m 5 of amyl nitrite For the lightning pains phenazone may be given Care should always be taken to see that no al urine is left in the bladder If so it should be removed daily heter If the urine is alkaline it is advisable to give acid sodium hate gr 60 at night to render it acid When there is hesitancy in rition tnc nuc vom m 10 to 15 t d s is of value A Charcot's should be kept at rest in a splint

## General Paralysis of the Insane

*(Dementia Paralytica)*

**efinition** A disease characterised by progressive deterioration mind with paralysis

to stand on one foot with his eyes shut, or to walk along a line with his eyes open, placing one foot in front of the other In cervical tabes the arms are chiefly affected and the patient cannot touch the tip of his nose with one finger with his eyes closed Pressure over the tendo Achillis (Abadie s sign) or over the ulnar nerve (Biernacki s sign) may not cause pain Optic atrophy may be an early sign, the discs being pearly white The external rectus muscles may be weak so that there is strabismus or diplopia and weakness of the levator palpebræ superioris may result in ptosis The tone of the leg muscles may be much diminished and the joint ligaments lax so that the knee can be hyperextended and the hip hyperflexed Trophic changes such as a perforating ulcer on the sole of the foot, or painless swellings of joints may occur The latter constitute Charcot's arthropathy Large joints such as the knee, hip, ankle elbow or shoulder, are usually affected The X ray of the joints may show atrophy or some hypertrophy of bone Later the patient may become very ataxic standing on a wide base and requiring two sticks to walk, and often throwing up the legs and bringing them down in a stamping manner heel first Still later he may become paralysed and bed ridden The blood The Wassermann reaction is positive in about 70% of early cases The cerebro spinal fluid Changes are found early in the disease and are of great diagnostic importance The Wassermann reaction is positive in about 70 to 90% of early cases The colloidal gold curve (Lange s test) gives a luetic response (see Fig 24) in about 85% of cases This is not diagnostic of tabes but differs from the paretic curve The cells are increased to about 80 per c mm , there being a pleocytosis in nearly 100% of cases Globulin is present (Nonne Apelt test) in about 90% of cases In a few active cases the spinal fluid is normal

**Differential Diagnosis** As the most hopeful results are to be expected if treatment is applied early, the diagnosis should not be delayed until the disease is firmly established as shown by absent knee jerks, an Argyll Robertson pupil and marked ataxia In the early stages peripheral neuritis due to diabetes mellitus or alcohol may be mistaken for tabes The urine should be tested for sugar in every case The pains in the limbs may be considered to be rheumatic The gastric crises closely simulate an acute abdominal lesion and if other signs of tabes are not looked for an unnecessary operation may be per formed A condition resembling the Argyll Robertson pupil may also be met with in encephalitis lethargica and in alcoholic neuritis but the pupils here are often neither small nor irregular and the reaction to accommodation is also defective

The tonic pupil with absent tendon reflexes was first described by Markus in 1905 In 1931 Adie drew attention to this condition and it is often known as the Adie syndrome The pupil change is unilateral in about 80% of cases women are more frequently affected than men the onset being generally in the third decade The reaction to light is usually completely absent although if the patient is kept in a dark room a sluggish reaction can sometimes be obtained The contraction of the pupil on accommodation is often somewhat sluggish but the

il becomes smaller than does the pupil in the other eye After xation of the accommodation effort re-dilatation is often slow the il taking several minutes to attain its original size Markus further ved that the pupil reacts to accommodation and not to convergence ankle jerks knee jerks and the arm jerks are absent in many cases cause is unknown

Difficulty in walking may occur in disseminated sclerosis but here plantar response is extensor and this also applies to Friedreich s ia In cerebellar ataxia there is usually nystagmus and the ataxy ot increased when the eyes are shut Syphilitic meningo myelitis closely simulate tabes but the course is usually more acute

**Course and Complications** The course is very variable and the ase may be arrested at any stage whereas an acute intercurrent ss may accelerate its progress In the elderly it tends to run a ign course Optic atrophy usually means that the patient will be d in a few years Complications include cystitis and pyelonephritis ocation of the hip may result from atrophy of the head of the femur eral paralysis of the insane sometimes occurs as a complication

**Prognosis** This is very variable Death may rapidly ensue the ient wasting and becoming bedridden or developing some com ation such as an intercurrent disease or infection of the urinary t In other cases the disease is arrested for long periods

**Treatment** A preliminary period of rest in bed for a few weeks is ays of value A course of treatment similar to that described on 07 for meningo vascular syphilis should be given Ataxia can be roved by having the soles of the shoes weighted and the patient can rained to regulate his movements by means of his eyes by Fraenkel s cises The patient practises placing his feet in certain positions and certain marks both when he is lying and on standing When the ase is arrested or burned out the cerebro spinal fluid usually ws an absence of globulin and no colloidal gold curve and the cell nt is low the Wassermann reaction is usually negative Gastric es may be treated by the subcutaneous injection of 1 ml of 1/1 000 enaline hydrochlor If relief is not obtained an intravenous injection ml of 10% calcium gluconate or of atropin sulph gr 1/100 may ve successful Laryngeal crises can generally be relieved by an lation of m 5 of amyl nitrite For the lightning pains phenazone 10 may be given Care should always be taken to see that no dual urine is left in the bladder If so it should be removed daily catheter If the urine is alkaline it is advisable to give acid sodium sphate gr 60 at night to render it acid When there is hesitancy in turition tnc nuc vom m 10 to 15 t d s is of value A Charcot s it should be kept at rest in a splint

## General Paralysis of the Insane

*(Dementia Paralytica)*

**Definition** A disease characterised by progressive deterioration the mind with paralysis

**Etiology** Dementia paralytica is caused by infection with the *Treponema pallidum* The infection is usually acquired, but congenital syphilis may give rise to juvenile general paralysis *Predisposing causes* 1 Age Usually between 80 and 50 years 2 Sex Males predominate Mental worry or brain work may also predispose

**Pathology** The body generally is wasted at autopsy The skull cap is thickened The dura mater is dense and hæmorrhagic pachymeningitis may be present The arachnoid is thickened and the pia mater is adherent at places to the brain so that on stripping it off, small portions of brain are removed with it The brain is atrophied the left hemisphere being more affected than the right in right handed people The convolutions are flat and the sulci wide but shallow The ventricles are dilated and the ependyma lining the floor of the fourth ventricle is granular and rough (*langue du chat*) The amount of cerebro spinal fluid is increased The grey matter is seen on section to be diminished Microscopically treponemes may be found, especially in the frontal poles The cortical pyramidal cells are degenerated There is a cortical perivascular infiltration with lymphocytes and plasma cells especially in the frontal lobes and to a lesser degree in the mid brain and cerebellum The neuroglial cells are proliferated Syphilitic lesions in other parts of the body are not often found

**Clinical Findings** A history of syphilitic infection about 10 or 15 *years previously may or may not be obtainable* *In the early stages* the patient may complain of headache or of a sensation of oppression on the top of the head, with insomnia Those who know him best will first detect alteration in his character such as lack of attention to details, failing memory passion emotion deterioration of judgment and intellect carelessness in habits or in dress and possibly laxity of morals Various delusions may occur, such as those of grandeur wealth excessive health, accomplishments or marked depression Epileptiform convulsions may occur or congestive attacks of an apoplectic type followed by hemiplegia or monoplegia which gradually pass off

*On Examination* The pupils are usually unequal and irregular and they often show the Argyll Robertson condition reacting to accommodation but not to light Tremors are seen in the lips and tongue The tone of the facial muscles diminishes, so that wrinkles disappear Tremors may also be seen in the hands The speech is altered so that it is hesitating or slurred and consonants such as the linguals and dentals are indistinct If the patient is asked to write the words are shaky and certain syllables are omitted Gradually the gait becomes unsteady and the legs are weak The tendon reflexes are exaggerated in the early stages but later the knee jerks and ankle jerks may be lost The plantar response is usually flexor but later it may become extensor *At this stage there is a spastic paresis* of the legs and the sphincter control of the bladder and rectum may be lost In the terminal stage the patient is bed ridden and trophic changes such as bedsores are likely to develop Some degree of optic atrophy is not uncommon The blood The Wassermann reaction is *positive in*

ly 100% of cases The cerebro spinal fluid The fluid is clear pressure is slightly increased The cells are increased to about ) 100 lymphocytes per c mm Large mononuclears or plasma cells also be present The Nonne Apelt test for globulin is positive Lange colloidal gold test gives a typical paretic curve (see Fig 24) Wassermann reaction is positive in about 95 to 100% of cases

**)ifferential Diagnosis** In the early stages general paralysis may mistaken for a functional disorder of the nervous system such as asthenia Cerebral syphilis is characterised by involvement of in of the cranial motor nerves In chronic alcoholic dementia pupils may not react to light and there are marked tremors but examination of the cerebro spinal fluid serves to differentiate A tal lobe tumour of the brain may also cause difficulty in diagnosis usually signs of increased intracranial pressure develop such as c neuritis or vomiting and the cerebro spinal fluid does not show changes characteristic of general paralysis

**Course and Complications** The course in an untreated case is gressive but remissions may occur In some instances signs of s also develop the disease then being known as tabo paresis

**Prognosis** If untreated death usually takes place within 4 years iagnosis If the disease is arrested by treatment in the early stages patient may be restored to health so that he is able to resume his k If arrest occurs in the later stages the patient closely resembles nimal with a very low grade intellect speech is difficult to under id he is childish and very emotional and requires constant attention

**Treatment** The results of penicillin treatment are better than se obtained with malaria The dosage of penicillin is as described meningo vascular syphilis (see p 407)

## DISEASES OF THE MOTOR NEURONES

### Motor Neurone Disease

**Introductory** A group of diseases is included under this heading h as Progressive ophthalmoplegia progressive bulbar paralysis gressive muscular atrophy amyotrophic lateral sclerosis and primary ral sclerosis In all of them the lesion consists of a chronic degenera i of the cells of the lower motor neurones situated in the pons medulla cord with changes of varying degree in the upper motor neurones ramidal tracts) There is an atrophic paralysis the degree of sticity present depending upon the extent of the lesion in the upper tor neurones This is most marked in amyotrophic lateral sclerosis l in primary lateral sclerosis

### Progressive Ophthalmoplegia

**Definition** A disease characterised by paralysis of the external ilo motor muscles due to degeneration of the nuclei of their lower tor neurones

**Etiology** The disease is often associated with syphilis It may

occur in tabes dorsalis, or in general paralysis of the insane or as a manifestation of progressive muscular atrophy

**Pathology** Degeneration occurs in the nuclei of the III, IV and VI cranial nerves

**Clinical Findings** The disease usually has an insidious onset the patient complaining of diplopia

*On Examination* There is often bilateral ptosis and weakness or paralysis of various external ocular muscles is found Later other bulbar nuclei are usually affected, death occurring as in progressive bulbar paralysis

## Progressive Bulbar Paralysis

### (*Labio glosso pharyngeal Paralysis*)

**Definition** A disease characterised by wasting of the muscles of the tongue, lips palate and pharynx, due to degeneration of the cells of origin of the lower motor neurones which supply them

**Etiology** The cause is unknown *Predisposing causes* 1 Age Usually over 50 2 Sex Males predominate slightly

**Pathology** There is atrophy of motor cells of certain cranial nerves, especially the X XI and XII and less often the V, VII and IX There is usually some degeneration of the pyramidal tracts The wasting is seen in the muscles of the tongue lips, palate, and in the muscles connected with the hyoid bone and the mandible The pharyngeal laryngeal and œsophageal muscles are only slightly affected

**Clinical Findings** The patient first experiences difficulty in speech, especially in pronouncing consonants, such as linguals and labials later disturbance of swallowing such as nasal regurgitation may occur or if the epiglottis is affected, swallowing will provoke coughing attacks Mastication may also be affected

*On Examination* When the disease is developed the sufferer from chronic bulbar paralysis is a pitiable object, the lower jaw hangs down saliva dribbles from the mouth and speech consists of an indistinct mumble The upper part of the face offers a surprising contrast being practically unaffected *The cranial nerves* I, II, III, IV, VI and VIII are normal V motor there is weakness of the muscles of mastication and of those attached to the hyoid bone V sensory, normal VII, weakness and wasting of the lip muscles the patient cannot whistle The orbicularis palpebrarum is only slightly affected IX, X and XI (accessory part) weakness of the palate is present and the vocal cord adductors may be affected XII wasting and paralysis of the intrinsic muscles of the tongue The tongue is shrivelled and wrinkled and lies paralysed on the floor of the mouth Fibrillary twitching is marked in the early stages If the pyramidal tracts are also involved the knee jerks are exaggerated The affected muscles are atonic there is no jaw jerk and the reaction of degeneration is present

*Varieties* A tonic atrophic variety of bulbar paralysis is described in which there is a combination of an upper and lower motor neurone lesion The tongue is paralysed but is smaller and firmer than normal A jaw jerk is present

*Spastic Bulbar Paralysis* (pseudo bulbar paralysis) This results ɪm a bilateral lesion of the upper motor neurones in the brain usually the internal capsule There is often a history of a previous attack hemiplegia The paralysed muscles are spastic, the tongue is ɪooth small and firm There is no reaction of degeneration and a w jerk is present

**Differential Diagnosis** Tumours vascular lesions or polio cephalitis of the mid brain may give rise to a rapidly developing ılbar palsy There are often sensory changes and the pyramidal ɪcts may be involved causing spastic lesions of the arms or legs . myasthenia gravis there is no atrophy of muscles ptosis is frequently esent and the muscles give the myasthenic reaction (see p 628) ɪphtheria may cause a transitory bulbar paralysis Basal syphilitic eningitis or a fusiform atheromatous dilatation of one vertebral tery may result in an unilateral bulbar palsv

**Course and Complications** The course is steadily progressive ɔmplications include inhalation bronchopneumonia or cardiac and spiratory failure

**Prognosis** Death usually occurs within two years from the onset

**Treatment** No cure is known Care should be taken in feeding mi solids are most suitable but in some cases resort must be made to ɪsal feeding

## Progressive Muscular Atrophy

### (*Progressive Spinal Muscular Atrophy*)

**Definition** A disease characterised by wasting of spinal muscles, ɪe to degeneration of the cells of origin of their lower motor neurones

**Etiology** The cause is not known In some cases there is evidence f syphilis as shown by a positive blood Wassermann reaction *'redisposing causes* 1 Age Usually between 25 and 40 years but may begin considerably earlier as at 12 years or later as at over ) years 2 Sex Males predominate

**Pathology** In the majority of cases the lesion is situated in the ɜrvical region of the cord but any level may be affected The cord ɜsion is a degeneration of the anterior horn cells with or without egeneration of the extramedullary anterior nerve roots In addition here is always some evidence pathologically of degeneration of the yramidal tracts and of the afferent spino cerebellar tracts arising in larke s column (Gowers and Flechsig s tracts) The muscles affected ɪow wasting of the fibres healthy fibres often lying side by side with ɪose which have degenerated

**Clinical Findings** In the typical cervical type the patient notices he gradual onset of weakness and wasting of muscles first in one hand sually the right and later in the other There may be aching or umbness in the hand

*On Examination* There is wasting of the small muscles of the hand hose of the thenar and hypothenar eminences the interossei and the ɪmbricals The hand assumes a claw shaped deformity with hyper xtension at the metacarpo phalangeal joints and flexion at the inter

phalangeal joints The thumb may rotate outwards to lie in the plane of the fingers Wasting spreads to the flexors of the forearms and then to the scapulo humeral muscles such as the deltoid and serratus magnus Certain muscles are typically spared such as the lower part of the pectoralis major, the triceps the latissimus dorsi and the upper part of the trapezius The neck muscles and intercostal muscles may be paralysed later Fibrillary contractions are seen early in muscles which are doomed to atrophy The deep reflexes connected with the affected muscles are abolished The electrical reactions show a *progressive lack of response both to faradisation and to galvanism* as the fibres degenerate, but often there is no true reaction of degeneration No sensory changes can be elicited Exaggerated knee jerks are an indication of a partial involvement of the pyramidal tracts

Other types of progressive muscular atrophy occur Thus in the shoulder type the scapulo humeral muscles are first affected and changes develop later in the hands In the neck type there is weakness first of the muscles which support the head so that it drops forward In the peroneal type there is weakness of the anterior tibial and peroneal muscles

**Differential Diagnosis** Other causes of wasting of the small muscles of the hand require consideration such as peripheral neuritis a cervical rib cervical pachymeningitis cervical caries, syringomyelia and *rheumatoid arthritis of the wrist The absence of sensory changes* and the presence of fibrillary contractions in progressive muscular atrophy are of great diagnostic value The shoulder type is differentiated from the facio scapulo humeral myopathy by the muscle groups affected and by the absence of fibrillary contractions in the myopathy The peroneal type is distinguished from peroneal muscular atrophy (Charcot Marie Tooth) by the fact that the latter occurs in children and there is often a familial incidence and there may be some sensory changes It is also diagnosed from the distal type of myopathy by the fibrillary contractions

***Course and Complications*** *If the onset is acute, then the course* is likely to be rapidly progressive In an average case the disease persists for 5 to 15 years Complications include the development of signs of amyotrophic lateral sclerosis or of chronic bulbar paralysis

**Prognosis** Death occurs in a variable time as described above

**Treatment** There is no cure known

### *Amyotrophic Lateral Sclerosis*

**Definition** A chronic disease of the brain and spinal cord with degeneration of the upper and lower motor neurones

***Etiology*** *The cause is unknown Predisposing causes* 1 Age Usually over 40 years 2 Sex Males predominate slightly

**Pathology** The upper motor neurone tracts in the cord degenerate the lesion appearing to extend upwards so that the motor cortical cells are affected The anterior horn cells in the cord degenerate, and certain cranial nerve nuclei may be similarly affected

**Clinical Findings** The disease usually starts insidiously with pains

umbness in the fingers hands or arms The patient then notices kness in the hands and arms with perhaps some stiffness in the legs
*On Examination* In an established case there is wasting of the ll muscles of the hands with deformity due to contractures (griffin s ) The muscles of the forearms arms and shoulders may also be ted There is weakness in the affected muscles and the neck may involved so that there is difficulty in supporting the head The scles show fibrillary twitching which can also be elicited by tapping r them Sensation is unaffected The tendon reflexes in the arms increased There is spasticity in the legs with hypertonus but the rer is retained and there is no wasting Later walking may become ossible The knee jerks and ankle jerks are increased there is le and patella clonus and the plantar response is usually extensor abdominal reflexes are increased in the early stages but later may be The muscles of the arms show a gradual failure of response both faradic and galvanic stimulation Sensation is not affected the incter control is normal A jaw jerk is often present due to eneration of the upper motor neurone fibres connecting with the tor nuclei controlling the jaw muscles (V motor) Affection of the nial nerve nuclei may be shown by disturbance of speech (dysarthria) sting of the facial muscles (orbicularis oris) difficulty in mastication l swallowing tremors and wasting of the tongue

**Differential Diagnosis** If in the early stages the hands or arms ne are involved the case closely simulates one of progressive muscular ophy to which the disease is allied and from which its separation somewhat artificial Other causes of wasting of the small hand scles must then be considered (see p 416) When the legs are marily affected the disease resembles primary lateral sclerosis h which it is also closely connected from the pathological standpoint he cranial motor nuclei are first involved the case presents the aspect bulbar paralysis In a fully developed case there is little difficulty establishing the diagnosis

**Course and Complications** The course may be rapid or com ratively slow Involvement of the cranial motor nuclei is a serious n Death often occurs from inhalation bronchopneumonia, due to erference with deglutition

**Prognosis** The disease is usually fatal within 4 years from its onset

**Treatment** This is usually only palliative Myanesin Elixir ½ fl t i d may be given for spasticity hot baths massage and passive ovements also tend to alleviate the spasticity

## THE SPINAL CORD

### Hæmatorrhacis

*(Meningeal Hæmorrhage)*

**Definition** Hæmorrhage into the meninges of the spinal cord

**Etiology** The hæmorrhage may be extradural or intradural xtradural hæmorrhage results from trauma or from rupture of an rtic aneurysm Intradural hæmorrhage (spinal subarachnoid hæmor

phalangeal joints The thumb may rotate outwards to lie in the plane of the fingers Wasting spreads to the flexors of the forearms and then to the scapulo humeral muscles, such as the deltoid and serratus magnus Certain muscles are typically spared such as the lower part of the pectoralis major the triceps the latissimus dorsi and the upper part of the trapezius The neck muscles and intercostal muscles may be paralysed later Fibrillary contractions are seen early in muscles which are doomed to atrophy The deep reflexes connected with the affected muscles are abolished The electrical reactions show a progressive lack of response both to faradisation and to galvanism as the fibres degenerate, but often there is no true reaction of degeneration No sensory changes can be elicited Exaggerated knee jerks are an indication of a partial involvement of the pyramidal tracts

Other types of progressive muscular atrophy occur Thus in the shoulder type the scapulo humeral muscles are first affected and changes develop later in the hands In the neck type there is weakness first of the muscles which support the head so that it drops forward In the peroneal type there is weakness of the anterior tibial and peroneal muscles

**Differential Diagnosis** Other causes of wasting of the small muscles of the hand require consideration, such as peripheral neuritis a cervical rib, cervical pachymeningitis, cervical caries, syringomyelia and rheumatoid arthritis of the wrist The absence of sensory changes and the presence of fibrillary contractions in progressive muscular atrophy are of great diagnostic value The shoulder type is differentiated from the facio scapulo humeral myopathy by the muscle groups affected, and by the absence of fibrillary contractions in the myopathy The peroneal type is distinguished from peroneal muscular atrophy (Charcot Marie Tooth) by the fact that the latter occurs in children and there is often a familial incidence and there may be some sensory changes It is also diagnosed from the distal type of myopathy by the fibrillary contractions

**Course and Complications** If the onset is acute, then the course is likely to be rapidly progressive In an average case the disease persists for 5 to 15 years Complications include the development of signs of amyotrophic lateral sclerosis or of chronic bulbar paralysis

**Prognosis** Death occurs in a variable time, as described above

**Treatment** There is no cure known

### Amyotrophic Lateral Sclerosis

**Definition** A chronic disease of the brain and spinal cord with degeneration of the upper and lower motor neurones

**Etiology** The cause is unknown *Predisposing causes* 1 Age Usually over 40 years 2 Sex Males predominate slightly

**Pathology** The upper motor neurone tracts in the cord degenerate the lesion appearing to extend upwards so that the motor cortical cells are affected The anterior horn cells in the cord degenerate, and certain cranial nerve nuclei may be similarly affected

**Clinical Findings** The disease usually starts insidiously with pains

control, exaggerated reflexes and an extensor plantar response while an atrophic paralysis develops in the hands and arms The Brown Séquard syndrome may also be found with loss of pain and temperature sensations on the side of the body opposite to the lesion and paralysis of the leg and trunk on the same side as the lesion in the cord If the bleeding is arrested a gradual improvement occurs with some residual lesions resulting in weakness of the hands or legs and dis sociated anæsthesia at various parts of the body Complications include cystitis and bed sores

**Prognosis** This is on the whole favourable as regards life unless the lesion is in the cervical region but some permanent disability is likely to persist

**Treatment** The patient must be kept absolutely at rest in bed and he may with advantage lie either prone or on his side An ice bag should be applied over the spine at the site of the lesion A subcutaneous injection of morphin sulph gr $\frac{1}{6}$ to $\frac{1}{4}$ tends to calm the patient and slow the circulation and is therefore of value

## Myelitis

**Definition** Inflammation and degeneration of the spinal cord

**Etiology** Acute myelitis may be met with under a variety of conditions Thus it may result from exposure to cold from trauma without visible external injury as after diving with the neck flexed or from exposure to the concussion of shells It may also be a mani festation of acute anterior poliomyelitis acute or subacute encephalo myelitis or of herpes zoster In some cases it occurs as a complication of illnesses such as influenza enteric fever dysentery measles syphilis or gonorrhœa Compression myelitis is due to pressure outside the cord and is considered separately Acute suppurative myelitis is a rare complication of bronchiectasis and infective endocarditis of tuberculous or suppurative spinal caries of purulent meningitis and of an infected meningocele or dermoid cyst Chronic myelitis is usually due to syphilis

**Pathology** In acute myelitis the cord is swollen at the affected site which may be located to a transverse area of the cord usually in the mid thoracic region more rarely there is a diffuse myelitis extending up and down the cord or disseminated patches are found On section the cord is soft and no distinction can be made between the grey and white matter On microscopical examination the nerve cells are found to have undergone chromatolysis and the nerve fibres are degenerated In chronic myelitis the cord is smaller than normal and pale

**Clinical Findings** *Acute Myelitis* The onset is usually com paratively sudden There may be a history of any of the antecedent conditions described above The patient is usually a young adult and he may notice a preliminary sense of numbness or tingling in the feet or legs or of pains in the legs or body or of a sense of girdle constriction round the body at the level of the lesion

*On Examination Thoracic Transverse Myelitis* The legs are found to be paralysed at the onset the paralysis is of the flaccid type with loss of the knee and ankle jerks The abdominal reflexes

rhage) may be due also to trauma or to rupture of a basilar or vertebral aneurysm In some cases it is due to convulsions, asphyxia purpura hæmophilia or to hæmorrhagic small pox It is a condition which is seldom encountered It is characterised by sudden onset and severe root pains and is often rapidly fatal

### Hæmatomyelia

**Definition** Hæmorrhage into the spinal cord

**Etiology** This is often due to an angioma or other vascular tumour of the cord, less often to hæmophilia *or to a syphilitic arteritis Small* hæmorrhages may be associated with purpura poliomyelitis syringo myelia, asphyxia tetanus and convulsions Trauma of the cervical *cord is not a common cause of hæmatomyelia Predisposing causes* 1 Age Usually between 15 and 30 years 2 Sex The lesion is more common in men

*Pathology The hæmorrhage often occurs in the cervical region of* the cord, the blood tends to collect in the grey matter especially the posterior commissure and in the anterior horn cells There is œdema around the hæmorrhagic area The hæmorrhage may extend up and down the cord for a few segments in a spindle shaped area It is absorbed if the patient survives leaving either a cyst or a scar The hæmorrhage thus interferes especially with the sensory tracts conveying pain and temperature sensations with the anterior horn cells causing paralysis of the atrophic type of the muscles supplied by the affected segments, and to a lesser degree with the tracts in the white matter such as the pyramidal and spino thalamic The posterior columns are usually spared

**Clinical Findings** There may be preliminary pains or numbness and tingling in the neck or shoulders The onset is usually sudden

*On Examination* (the cervical type) The patient is conscious There is paralysis of the arms and legs of a flaccid type at the onset with loss of deep reflexes, constipation and retention of urine In some cases the paralysis is very slight, but there is dissociated anæsthesia with loss of temperature and pain sensations in the hands and arms A band of hyperæsthesia may be detected at times at the site of the level of the lesion There is no disturbance of joint, muscle vibration or touch sensations In a severe case there is paralysis of the abdominal and intercostal muscles and respiration is carried on by the diaphragm The cervical sympathetic may also be paralysed as in syringomyelia The temperature is usually normal or subnormal at the onset The cerebro spinal fluid This is normal at the onset but later may be yellow (xanthochromia) from blood pigments

**Differential Diagnosis** Hæmatomyelia must be differentiated from acute transverse myelitis and from syringomyelia The onset is more gradual in syringomyelia and also in myelitis and in the latter the temperature is raised

**Course and Complications** If the hæmorrhage spreads death may occur rapidly from respiratory failure In other cases after about a week the lower limbs may show a spastic paraplegia with loss of sphincter

spirits and the heels allowed to rest in rings All crumbs must be removed from the bed The posture should also frequently be changed The bowels should be emptied every other day with an enema if necessary and a watch kept on the bladder If there is retention of urine it may be possible to empty the bladder by suprapubic pressure otherwise catheterisation every 8 hours is required Alternatively an apparatus can be used to produce automatic tidal drainage of the bladder The urine should be kept acid by the administration of acid sod phosphate gr 30 to 60 twice a day If the Wassermann reaction is positive a course of anti syphilitic treatment should be given (see p 407)

## Compression Myelitis

**Etiology** In the majority of cases the compression of the cord is produced gradually It may be due to affections of the vertebræ such as tuberculous or syphilitic caries Paget s disease vertebral tumours such as primary or secondary sarcoma and secondary carcinoma an exostosis osteoma or chondroma It may also occur as a complication of rheumatoid spondylitis or be due to protrusion of an intervertebral disc Tumours of the meninges or nerve roots may compress the cord such as an extradural sarcoma or an intradural sarcoma or fibroma Leukæmia and Hodgkin s disease are comparatively rare causes Syphilitic pachymeningitis hypertrophica may compress the cord usually in the cervical region A hydatid cyst or a simple meningeal cyst will cause pressure It is rare for an aortic aneurysm to erode the spine sufficiently deeply to compress the cord With a fracture dislocation of the spine the compression is rapidly produced This is a surgical condition

**Clinical Findings** *Affections of the Vertebræ* The patient is often a child if the lesion is tuberculous caries and in other cases he is generally an adult Pain is complained of in the back and often very severe root pains are present radiating in a segmental distribution round the body or along the limbs due to posterior nerve root involvement Slight movement of the spine or coughing may cause very severe agony Later weakness with spasticity occurs in the legs and numbness or tingling

*On Examination* In caries angular curvature of the spine is often seen and local tenderness on tapping may be found over one or more vertebræ In the early stages a zone of hyperæsthesia may be present corresponding with a segmental distribution of the cord Later there may be anæsthesia over an area in which severe pain is felt (anæsthesia dolorosa) Motor signs sometimes occur before the sensory ones and when present resemble those described above for transverse myelitis there being spastic weakness of the legs exaggerated deep reflexes and an extensor plantar response With progressive pressure the extensor muscles are first affected the legs being spastic and extended and from time to time involuntary clonic contractions occur (paraplegia in extension) Stimulation of the sole of the foot may cause not only extension of the big toe (dorsi flexion) but also flexion of the knee and hip Later the extensors lose their tone and the flexors are con

are lost but the epigastric reflex is preserved if the lesion is below the 9th thoracic segment Priapism may be present There is retention of urine and of fæces Sensation is lost over the legs and lower part of the body, but at the level of the lesion a zone of hyperæsthesia may be found Later the paralysis becomes spastic the extensor muscles being affected more than the flexors the deep reflexes of the legs are exaggerated and the plantar responses become extensor

*Low Cervical Transverse Myelitis* This is more rare The arms and legs are paralysed, and later, while the legs become spastic the paralysis of the arms is of the flaccid atrophic type The cervical sympathetic may be involved on one or both sides, with enophthalmos and constriction of the pupil Sensation is lost below the level of the lesion

*Lumbar Myelitis* There is flaccid paralysis of the legs, with loss of the knee and ankle jerks and of the plantar responses The latter may at times be extensor The bladder and rectum become incontinent The temperature is raised in cases due to infection

The cerebro spinal fluid In any type this may show an increase of globulin and of cells

*Acute Diffuse Myelitis* There is an ascending paralysis which spreads up the legs to the trunk and arms, together with an ascending anæsthesia There is loss of sphincter control Bed sores and cystitis are very liable to develop

*Chronic Myelitis* The paralysis is of gradual onset with first weakness and stiffness in the legs and later sensory loss exaggeration of the deep reflexes and extensor plantar responses

**Differential Diagnosis** In spinal thrombosis or hæmorrhage the onset of the paralysis is generally more rapid than in myelitis and there is no fever In acute infective polyneuritis there is a flaccid paralysis pains in the legs are usually present there is no loss of sphincter control and the sensory disturbance is less marked than in myelitis Landry s paralysis is by some considered to be indistinguishable from acute infective polyneuritis In hysterical paraplegia other stigmata of hysteria can usually be detected

**Course and Complications** The course in acute myelitis is very variable in the diffuse variety the lesion may rapidly spread up the cord and death ensue from paralysis of the muscles of respiration In transverse myelitis there is a tendency to recovery which is however rarely complete Some degree of spastic weakness of the legs is liable to persist with contractures of the flexor muscles so that the patient has to walk with the aid of two sticks Impotence is liable to occur as a sequela Complications include cystitis pyelitis, pyelonephritis and bed sores

**Prognosis** In the diffuse and high cervical type death usually occurs in a day or so, in other cases the prognosis is as described above

**Treatment** The patient should be nursed on a water bed and great attention paid to the skin to prevent the formation of bed sores The skin must be kept very clean, thoroughly dried, powdered with starch and zinc oxide pressure points must be rubbed with methylated

thoracic region the spinal cord segments 1 to 9 lie two spines higher and from the Th 10 downwards the spinal cord segments are three spines higher The level of the cord lesion can also be more roughly determined by the presence or absence of the superficial and deep reflexes which correspond with certain spinal levels as enumerated on p 299 The superficial reflexes are abolished below the level of the lesion whereas the deep reflexes are exaggerated The various muscles also correspond with certain spinal levels as shown in the table on p 299 and so paralysis of definite muscles indicates the site of the lesion Myelography will indicate the level of obstruction in a spinal cord compression due either to intra or extra dural causes

**Differential Diagnosis** In diagnosing the cause of spinal compression the spine should first be examined for deformity and also a radiogram taken If the disease is due to an affection of the vertebræ it can usually be determined in this way The presence of a primary malignant growth elsewhere in the body and of severe root pains is very suggestive of a malignant deposit in the vertebræ In meningeal tumours the diagnostic features are the root pains and subsequent spastic paralysis at first unilateral and later bilateral with sensory disturbances It is not usually possible to differentiate clinically between a meningeal and spinal tumour When pain is the most prominent symptom a diagnosis of rheumatism or neuritis is liable to be made unless a careful examination is carried out to exclude tumours or compression myelitis

**Course and Complications** The course depends upon the cause of the compression Thus in malignant growths of the vertebræ the course is rapidly progressive whereas a meningeal tumour may exist for several years In spinal caries the course is very variable and often the disease is arrested

**Prognosis** This must vary with the cause as described above

**Treatment** Spinal caries is treated by streptomycin isoniazid para aminosalicylic acid rest and exposure to sunlight In secondary malignant vertebral tumours nothing can be done to cure but pain may be relieved by division of the posterior nerve roots Many spinal and meningeal tumours can be removed by operation The Wassermann reaction should always be carried out and if positive a course of anti syphilitic treatment given before an operation is performed If there is no improvement operation should not be delayed for more than 3 to 4 weeks as a tumour may be present which is not a gumma

### Compression of the Cauda Equina

**Etiology** This may be due to a fracture dislocation in the region of the lower lumbar vertebræ to spinal bifida chronic arachnoiditis a fibrous band a prolapsed intervertebral disc or to a tumour Extra dural tumours include a sarcoma and chondroma and intradural tumours a meningeal or perineural fibroblastoma The cauda equina embraces the nerve roots below those arising from the second lumbar segment of the cord

**Clinical Findings** The patient complains of pain in the lower part of the back or the legs having a root distribution There is also

tracted (paraplegia in flexion) the hips and knees being flexed and the knee and ankle jerks being lost With malignant tumours of the vertebræ excruciating root *pains are a characteristic feature*, there is localised vertebral tenderness but little liability to motor weakness as death usually occurs before there is time for this to develop

*Tumours of the Meninges and Spinal Cord* These are usually intradural *They may be extramedullary arising in the meninges or* nerve roots or intramedullary from the cord The former include a sarcoma endothelioma myxoma lipoma meningioma, neurofibroma neuroma gumma psammoma or hydatid cyst and the latter a glioma *hæmangioma* tuberculoma sarcoma and rarely a gumma The patient usually first complains of pain in one side of the back or along a limb in an area corresponding with one posterior nerve root later weakness appears in the leg or arm on the side of the lesion The pain is often worse on coughing sneezing or at night With an intramedullary tumour paralysis and anæsthesia are usually present before the occurrence of pain

*On Examination* There may be spastic weakness with exaggerated reflexes and loss of postural sensation in the limb on the same side as the lesion and sensory changes with loss of pain temperature and touch on the other side of the body The sensory changes extend up to the level of the lesion The cutaneous reflexes are abolished below the lesion and are normal above it In a later stage both legs may be affected with spastic weakness and the sensory loss is also bilateral there is also loss of sphincter control The cerebro spinal fluid In compression myelitis this shows the characteristic changes known as *the loculation syndrome* there being an increase of protein up to 3 or 4% (normal 0 02%) below the obstruction and a yellowish tinge is seen on looking down through a column of the fluid in a tube This is due to extravasated blood pigment and is called xanthochromia Cells are absent or not in excess The fluid also often clots spontaneously In cases of spinal cord tumours it is necessary to localise the site of the lesion accurately, so that the surgeon shall know at what level to operate This is done by a consideration of the sensory motor and reflex changes The lesion is usually situated one or two segments above the level indicated by the sensory loss (see Fig 22 for segmental areas of posterior nerve roots) as the sensory fibres run up in the cord obliquely for a short distance before crossing Further as the fibres conveying pain and temperature sensation decussate before those of touch with a lesion in the cord at a definite site the cutaneous area showing the loss of touch will be lower than that showing loss of pain and temperature The higher up the cord the longer distance the sensory fibres travel before they decussate so that in the upper cervical region the lesion may be situated four or five segments above the segmental level of loss of sensation in the body Further the spines of the vertebræ do not lie exactly over the corresponding spinal cord segments This must be borne in mind in marking out the level over the spine for exposing the cord at operation In the cervical region the spinal cord segment lies at a level of one spine higher in the

present in the grey matter of the spinal cord and brain The grey matter of the anterior horns of the cervical and lumbar enlargements is especially affected The changes in some of the anterior horn cells appear to be reversible as shown by clinical recovery at times of certain paralysed muscles There is inflammation of the leptomeninges particularly over the anterior fissure of the spinal cord. The grey matter of the cord on section may appear swollen and pink Microscopically there is a round-celled infiltration around the anterior spinal vessels many of the cells being polymorphonuclears producing a 'cuffing' effect There is degeneration of cells in the anterior horns The cerebral and cerebellar cortex the basal ganglia and brain stem may also be affected Sensory changes are probably due to involvement of dorsal root ganglia There may also be a generalised enlargement of lymphatic glands with parenchymatous changes in the liver and kidneys

**Incubation Period.** This is from 4 to 30 days or longer

**Clinical Findings** *Subclinical infection* may occur the individual being infected with the virus and developing immunity without there being any symptoms of ill health The *minor illness* is more common in children than in adults It is characterised by fever headache vomiting or sore throat After lasting for a day or so it may abort constituting the whole illness In other cases it is followed after an interval of about 2 days by the *major illness* or the major illness may appear without a preliminary minor illness The major illness may be paralytic or non paralytic

*Non paralytic cases* fortunately constitute the majority of cases in many epidemics the infection stopping before the anterior horn cells or motor nuclei are involved The symptoms in these cases belong to the *prodromal or pre-paralytic stage* They are as follows The patient is taken ill comparatively suddenly with headache malaise pains in the neck back and limbs nausea vomiting diarrhœa and perhaps convulsions The temperature is raised to about 103 F In some instances there is diplopia This prodromal stage may be very short or it may last for a day or so Signs of meningeal irritation may or may not then develop There is very intense headache

*On Examination* Definite neck rigidity may be found If Kernig s sign is not present the spinal sign may be elicited The patient lies on his side if now an attempt is made by the examiner to flex the spine the movement is resisted by the patient owing to pain in the back The skin often has a pink hue and there is an anxious look in the eyes Small and uncontrolled movements may be seen in the fingers and hands The cerebro spinal fluid The fluid is clear and under increased pressure There is an excess of cells present (15 to 200 per c mm.) both polymorphonuclears and lymphocytes The protein is increased, the sugar and chlorides are normal and no organisms are found. The blood There is often a leucocytosis of about 25 000 per c.mm

*The Paralytic Stage* Muscular paralysis is noted about the second or third day It is usually maximal at the onset, but in some cases spreads rapidly Various types of disease are described of which the commonest

weakness of one or both legs, and often retention or incontinence of urine and fæces

*On Examination* The leg or legs show muscular weakness or paralysis, with loss of tone and wasting The knee jerk and ankle jerk are absent The plantar response is absent or flexor Loss of sensation is found in the legs and perineum

**Prognosis** This varies with the cause when due to trauma recovery often occurs

**Treatment** If a tumour is suspected an exploratory laminectomy should be performed Otherwise the treatment is as for compression myelitis

### Acute Poliomyelitis and Polioencephalitis
### (*Heine Medin Disease Infantile Paralysis*)

**Definition** An acute disease characterised by lesions having a special affinity for the grey matter of the anterior horns of the spinal cord and for the motor nuclei of the brain stem

**Etiology** The disease is caused by a virus of which there are 3 types, Brunhilde, Lansing and Leon Infection takes place either by droplets disseminated by carriers or abortive cases, or by milk, food or water contaminated by fæces or flies Epidemic and sporadic infection occurs It is now recognised that spread of infection in the family or in hospital is very liable to occur *Predisposing causes* 1 Age Chiefly between 2 and 5 years adolescents and young adults are sometimes affected Since 1947 the majority of cases in Great Britain have developed between the ages of 6 to 12 years 2 Sex Males predominate slightly 3 Season Summer and autumn but in all districts sporadic cases are constantly present 4 Locality America Canada Australia Scandinavia and to a lesser degree Great Britain 5 Tonsillectomy This predisposes to infection and the bulbar type is then liable to occur 6 There appears to be a relationship between immunisation against pertussis and diphtheria and the development of poliomyelitis within the next 30 days Further, the severe paralysis is likely to be localised to the limb last inoculated Such cases have been observed in Australia and Great Britain

**Pathology** The virus is present in the naso pharyngeal mucous membrane in carriers convalescents and in acute cases It is also found in the fæces It is believed that in the majority of cases infection in man is through the alimentary tract the virus spreading to the central nervous system along autonomic nerves Less often infection is through the naso pharynx especially after tonsillectomy The virus may be present in the fæces during convalescence for over 3 months and it may also be detected in sewage Flies fed on infected human fæces can convey the virus to food

In human beings the virus is found in the nervous system in the motor cortex diencephalon mesencephalon medulla pons and cord and in the alimentary system in the pharyngeal region in the wall and contents of the ileum and in the contents of the colon It is not found in the blood cerebro spinal fluid or viscera Destructive lesions are

paralysis has appeared and the muscles are tender and painful the disease may be mistaken in infants for scurvy acute rheumatism osteomyelitis or syphilitic epiphysitis The neuritic form is differentiated from multiple neuritis by the fever and by special groups of muscles being affected The spreading type closely resembles Landry s paralysis

**Course and Complications** Although in the majority of cases the paralysis is maximal at the outset the paralysis may spread in a few hours from muscle group to muscle group Usually many of the anterior horn cells which are put out of action at the onset recover with corresponding improvement in muscle power noticeable after about 7 to 10 days Relapses are not unknown during the course of the illness with rise of temperature and further paralysis of muscles but they are not usually serious Some of the affected muscles recover completely others undergo partial recuperation and others may be permanently paralysed Scoliosis may result from a minor attack of poliomyelitis which has picked out certain spinal muscles The bones of the affected limbs do not usually grow as well as normal with subsequent shortening of the leg or arm Contractions and deformities result from permanently damaged muscles Progressive muscular atrophy has occasionally been noted as a late sequela

**Prognosis** The mortality rate in an epidemic varies between about 10 and 20% In the bulbar type the mortality rate varies from 40% to 80% Complete recovery may be expected even if all four limbs are paralysed if the superficial and deep reflexes persist Muscles which are painful and tender are more likely to recover than those which are insensitive The mortality is very high when the medulla is affected and spreading infection is usually very serious death resulting from bulbar or spinal respiratory paralysis or from bronchopneumonia A muscle which responds to faradisation usually recovers but the prognosis is serious if the reaction of degeneration is present Some degree of recovery may be expected for a year after the onset of the disease

**Treatment** *Prophylactic* The Salk trivalent formalinised vaccine or modifications of it are on trial Two intramuscular injections of 1 ml each are given at intervals of 4 weeks and a third injection may be given 7 months later Unvaccinated contacts may be given an intramuscular injection of concentrated immune globulin (gamma globulin) 0 1 ml /lb body weight or of 10 ml of convalescent serum obtained from a patient within 10 days of his becoming afebrile Food milk and water should be protected from flies and flies destroyed by D D T If an outbreak occurs in a boarding school there are arguments for and against sending the pupils home The risk of so doing consists in planting a carrier in a new zone where he will spread the infection The child must be kept in quarantine for 21 days On the other hand if healthy children are kept in the school they run the risk of contracting the disease from a carrier or from infected food or water

*Curative* The patient should be isolated for three weeks after the temperature has become normal or for 6 weeks from the clinical onset

is the spinal variety The legs are more often affected than the arms The patient complains of pains in certain muscles with paralyses

*On Examination* Certain muscles are in a state of flaccid paralysis There is a lower motor neurone lesion of a segmental character The deep reflexes are generally sluggish or lost, the abdominal reflexes are abolished and sensation is normal The muscles may be unduly tender on pressure or on joint movement The muscles especially affected in the leg are the dorsiflexors of the ankle and the extensors of the hip and knee In some cases one arm and the other leg are affected, but all four limbs may be paralysed Spasm is often found in the back of the neck the erector spinae and hamstring muscles A maculo papular eruption may be seen on the affected limb The bowels are usually constipated If the lesion is situated in the mid thoracic region there may be a definite transverse myelitis, with sphincter paralysis, flaccid paralysis below the level of the lesion and loss of deep reflexes The flaccidity is later followed by a spastic paraplegia

*Varieties* In addition to the spinal type described above, certain other varieties of the disease are recognised *The brain stem type* This is the most important The VII, VI and III nerves are most commonly affected In some cases the lesions are bulbar, there being dysphagia, dysarthria dysphonia disturbances of breathing and irregularity of the heart Bulbar poliomyelitis has been frequently reported following tonsillectomy performed during epidemics of polio myelitis *The neuritic type* There are severe pains in the arms or legs The deep reflexes are diminished There is no paralysis *The spreading type* This resembles Landry s paralysis The paralysis may spread up or down the cord in the space of a few hours and if the respiratory muscles are all paralysed death results *The cerebellar type* The patient has intense headache vertigo, vomiting on moving the head, ataxia of speech and of spinal muscles and nystagmus *The cerebral type* The onset is with generalised convulsions persisting for several hours, followed by hemiplegia or paraplegia with spasticity and an extensor plantar response There may also be mental deterioration and athetotic or choreic movements *Mixed types* A combination of cere bral and spinal lesions may be present Thus one leg may show a spastic and the other a flaccid paralysis

**Differential Diagnosis** Sporadic cases may be difficult to diagnose at the onset There are no distinguishing features during the prodromal period In the meningeal stage the cerebro spinal fluid usually shows the characteristic findings described above In tuberculous meningitis the sugar content and then the chloride content are reduced, in bacterial meningitis the sugar content is lowered and the causative organism is present and in encephalitis lethargica polymorphonuclear cells are not usually found In encephalitis lethargica paralyses are not so common and usually not so acute in onset and meningeal symptoms are rare In polioencephalitis the onset is usually more acute more than one member of a family is likely to be affected the patient is not usually over the age of 30 the temperature is generally higher, and the course of the acute stage of the disease is shorter than in encephalitis lethargica When

weakness first in one leg and then in the other There may be prodromal symptoms such as malaise numbness or tingling in the feet or pains in the back or legs The paralysis rapidly spreads up the legs In the case originally described by Landry the shoulders, the arms and hands were next paralysed and finally paræsthesia spread to the trunk and there was dysphagia This corresponds with a neuritic origin of the disease In other cases subsequently classified as Landry's paralysis the trunk is affected after the legs then the arms neck head and finally the tongue and muscles of deglutition Involvement of the diaphragm and intercostal muscles usually results in death from respiratory failure.

*On Examination* Spinal motor system There is flaccid paralysis of the legs trunk and arms and finally of the neck face and head The diaphragm and intercostal muscles may or may not be affected The muscles do not waste or only very slightly Spinal sensory system Often there are no changes some loss of touch sensation may be detected in the toes The reflexes The deep and superficial reflexes are lost The sphincters are usually unaffected but there is often retention of urine and of fæces from lack of muscular power In some cases the XII XI VII or III IV and VI nerves may be affected. Trophic changes These do not usually occur Electrical reactions There is no reaction of degeneration Response to galvanism is usually lost but faradic response remains Lumbar puncture The fluid is usually under increased tension It is clear and may contain an excess of protein and of cells Cerebration The mind usually remains clear until the end. The temperature is not usually raised. The spleen may be palpable

**Differential Diagnosis** Acute toxic polyneuritis By some authorities this is held to be indistinguishable from Landry s paralysis However in the former there is more likely to be fever pain in the limbs with anæsthesia and wasting of muscles The abdominal reflexes may also be present Acute ascending myelitis Here there are more marked sensory changes with sphincter paralysis and the development of bed sores The plantar responses are likely to be extensor Acute polioencephalomyelitis The spread of the paralysis is more irregular there is fever and wasting of the paralysed muscles An acute ascending paralysis may occur in rabies and in porphyria

**Course and Complications** The disease is usually rapidly progressive Deglutition bronchopneumonia may occur as a complication

**Prognosis** Death frequently occurs in a few hours or days This may be due to paralysis of the respiratory muscles or of the heart or to deglutition bronchopneumonia If the patient recovers there is usually no wasting or paresis of the affected muscles

**Treatment** When the respiratory muscles are affected the patient should be put in a respirator and hypodermic injection of strychnin hydrochlor gr 1/60 and atropin sulph gr 1/120 given four hourly Nasal feeding and inhalations of oxygen may also be required Lumbar puncture should be made as required to relieve the increased tension of the spinal fluid

of the disease As the virus is eliminated in the fæces and probably in the urine, strict precautions should be taken with regard to their disinfection and disposal The patient should be kept in bed even in the mildest case, as this is likely to prevent the onset of the paralytic stage The general consensus of opinion is opposed to the use of convalescent or adult serum Lumbar puncture during the meningitic stage usually relieves temperature, headache and muscular pains, and may be repeated for the first 2 or 3 days Great care must be taken to prevent stretching of paralysed muscles and a footboard should be used to maintain dorsiflexion of the feet and to take the weight of the bed clothes The limbs must be placed in such a position that the affected muscles are relaxed and the position maintained by pillows sandbags, or celluloid splints Gentle passive movements may be used with benefit in the early stages The tightness or spasm of the spinal or hamstring muscles may be relieved by hot packs applied for 1 to 2 hours morning and afternoon, as advised by Elizabeth Kenny Patients who sweat profusely should be given plenty of fluid by mouth and sodium chlorid gr 7½ to 15 t d s When the respiratory muscles are affected, artificial respiration may be carried out for prolonged periods automatically by the use of a respirator either of the cuirass or box type In the latter the whole patient except the head is enclosed For general use it is preferable Pain may be relieved by the administration of aspirin in doses of gr 5 to 10 t d s according to the age of the patient Priscol may be given for relief of pain and to increase the vascularity of the affected muscles The dose for an adult is two mg 25 tablets four times a day The muscles should not be massaged during the period of active infection and no electrical treatment should be given After 3 or 4 weeks gentle massage may be applied Orthopædic treatment may improve contractures or deformities, but will not be required for at least a year after the onset of the illness

## Landry's Paralysis

### (*Acute Ascending Paralysis*)

**Definition** A disease characterised by acute ascending paralysis *with little or no sensory or sphincter disturbance*

**Etiology** The cause is not known The disease is probably due to a toxin By some authorities Landry s paralysis is regarded as a manifestation of acute toxic polyneuritis and by others as an acute polioencephalomyelitis Probably both types exist One case of acute ascending myelitis has been shown to be due to a neurotropic filtrable virus *Predisposing causes* 1 Age 20 to 45 years 2 Sex Males predominate

**Pathology** In some cases no changes are found post mortem, in others there is hyperæmia of the vessels of the cord, chromatolysis of the anterior horn cells or interstitial changes in the peripheral nerves

**Clinical Findings** The patient is usually suddenly seized with

muscles The knee jerks and ankle jerks are brisk and ankle clonus and patella clonus may be present The plantar response is extensor The abdominal and cremasteric reflexes are lost Areas of loss of cutaneous sensation may be found on the legs the bone vibration sense is diminished or lost in the legs joint sensation is usually lost Pallor may be detected in the temporal halves of the optic discs In cases of loss of vision in one eye there is a retrobulbar neuritis The affected pupil is usually dilated and does not respond well to direct stimulation by light although it contracts consensually when a light is shone into the other eye A small central scotoma for colours may also be present Weakness of accommodation may be a troublesome feature A history of transitory diplopia or urinary disturbance may be obtained on questioning In more advanced cases the triad of symptoms described by Charcot may be found These are nystagmus intention tremors and scanning speech The nystagmus is a fine horizontal one Jelly nystagmus is almost a diagnostic sign when present On ophthalmoscopic examination the fundus appears to quiver rapidly The intention tremors are seen when the patient is asked to lift up a glass of water or perform some other movement and become more marked towards the completion of the act The speech is slow and the syllables are pronounced somewhat abruptly There may be difficulty in beginning micturition or incontinence In the later stages contractures occur in the spastic muscles and there are marked tremors of the head neck and arms so that the patient is bed ridden and unable to look after herself Other types include a cerebellar variety characterised by vertigo ataxia and nystagmus and a cerebral one with progressive hemiplegia The blood The Wassermann reaction is negative The cerebro spinal fluid This may be normal In about 80 to 70% of cases a paretic gold curve is obtained (see Fig 24) There may also be an excess of cells and globulin

**Differential Diagnosis** In the early stages the disease is very liable to be mistaken for hysteria or the retrobulbar neuritis may be thought to be due to sphenoidal sinusitis When there is spastic paraplegia spinal syphilis must be excluded and local pressure due to bony changes or a cord tumour The ataxy must be distinguished from that due to Friedreich s ataxia in which the deep reflexes are depressed and ataxic paraplegia raises the consideration of subacute degeneration of the cord

**Course and Complications** In the early stages remissions are a very characteristic feature, the weakness of the limb disappearing and reappearing later Fresh symptoms appear at different parts of the body as new lesions develop in the central nervous system Arrest may occur at any stage or the patient may rapidly become bed ridden Complications include septic infection of the urinary tract and inter current lung infections

Prognosis This is unfavourable death sometimes occurring in a few months but in other cases not for 30 years or more after the onset

Treatment There is no known cure Any obvious septic focus should be eradicated A course of neoarsphenamine may be given

## Disseminated Sclerosis

*(Multiple Sclerosis)*

**Definition** A disease characterised by spastic weakness of muscles, with a progressive course often interrupted in the early stages by remissions

**Etiology** The cause is not known It has been thought to be due to the action of toxins or to the presence of a myelinolytic ferment in the blood Various possible causative organisms have been described without sufficient evidence to justify their acceptance Other causes suggested include scattered venous thrombosis with increased coagulability of the blood localised areas of vasoconstriction and allergic reactions *Predisposing causes* 1 Age Usually between 20 and 40 rarely under 16 or over 45 2 Sex Females predominate slightly There is no familial incidence but several cases may occur in a household The disease may show itself after an attack of influenza scarlet fever or exposure to cold It is a very commonly occurring nervous disease

**Pathology** Lesions are scattered throughout the brain and spinal cord, varying in size from a pin point or less to about ½ inch in diameter The recent lesions are pinkish in colour and the old ones greyish white They are present both in the grey and white matter of the central nervous system and the optic nerve is often affected There is degeneration of the myelin sheath of the nerve fibres the axis cylinder usually remaining intact There is also a perivascular infiltration with lymphocytes and plasma cells and some local œdema In the older lesions there is proliferation of neuroglial tissue Secondary degeneration above or below the damaged area in the cord rarely occurs

**Clinical Findings** In the majority of cases the disease has an insidious onset, although the first symptom noted occurs suddenly The patient is a young adult who may notice weakness in one leg on walking the foot tending to catch in the ground or there may be numbness or tingling in a leg or vague aching Early urinary symptoms may occur such as difficulty in beginning micturition or retention In other cases the patient suddenly loses the vision in one eye recovery taking place in a week or so, or there may be a transient attack of double vision An attack of acute unilateral retrobulbar neuritis lasting a week or so may precede the onset of other symptoms by several years *The acute variety* Rarely the disease begins with an attack of hemiplegia during which the patient does not lose consciousness or with epileptiform convulsions It is unusual for the weakness to affect the arms before the legs

*On Examination* In the early stages no abnormal signs may be found, and in such a case the patient should be re examined from time to time so that a diagnosis can be established as soon as possible A tendency to laugh easily unnatural cheerfulness or euphoria may be noted in the early and advanced stages of the disease The signs which occur in disseminated sclerosis are as follows Weakness may be detected in certain groups of muscles such as the flexors of the ankles or of the hip There is a tendency to spasticity of the leg

from the posterior root on one or both sides which convey impulses of pain and temperature and cross the cord in the anterior commissure to ascend in the spino thalamic tract on the other side If the cavity extends further outwards it may involve the fibres entering from the posterior root which convey impulses of touch and ascend on the same side of the cord Extension forwards into the grey matter causes atrophy of the anterior horn cells and lateral extension may interfere with the pyramidal tract

**Clinical Findings** The onset is usually insidious The patient may first notice pains in the hand or arm or weakness of the hands or clumsiness in fine movements In other cases he burns his fingers with a cigarette without feeling heat or pain or a cut on the finger is painless If there is gross dilatation of the cervical cord severe pain may be felt in the neck

*On Examination* In an established case of the cervical type the following changes may be found There is usually kyphoscoliosis in the thoracic region and a spina bifida may be present Spinal motor system There is wasting of the small muscles of one or both hands so that a claw hand or monkey hand is seen The muscle wasting may extend to the arm and shoulder girdle with winging of the scapula In muscles which are actively wasting fibrillary contraction may be seen The deep reflexes of the arms may be increased or diminished and a reaction of degeneration may be present in the affected muscles There is often spastic weakness of the legs with increased knee jerks and ankle jerks and an extensor plantar response Spinal sensory system Sharply defined areas of dissociated anæsthesia are present on the hands arms neck trunk etc There is loss of sensation to heat cold and pain whereas touch sensation the discrimination of two points joint sensation muscle sensation and the vibration sensation are preserved In some cases there is a complete hemi anæsthesia to pain and temperature senses on the opposite side of the body below the level of the lesion due to involvement of the spino thalamic tract Trophic changes include painless swelling of the shoulder elbow or wrist similar to the Charcot s arthropathy of tabes dorsalis The bones of the arm may break spontaneously The hands may be enlarged and thick *main succulente* owing to thickening of the subcutaneous tissues painless ulcers or subcutaneous whitlows may occur on the fingers patches of red or blue skin may be seen on the hands Involvement of the cervical sympathetic on one side is indicated by Horner s syndrome *i e* enophthalmos narrowing of the palpebral fissure contraction of the pupil and absence of sweating on one half of the face Other types of the disease are more rarely met with They include *The thoracico lumbar variety* There is wasting of the muscles of the pelvic girdle and legs and dissociated anæsthesia of the legs *The sacro lumbar variety* The small muscles of the feet and the leg muscles waste Trophic lesions may be seen on the feet There is dissociated anæsthesia of the feet or legs There is usually loss of control of the bladder and rectum The deep reflexes are often exaggerated in the legs and the plantar response is extensor

starting with 0 1 G, and increasing to 0 6 G, six intravenous injections in all The course may be repeated every 6 months Other treatments which have been tried and found wanting include fever therapy, vasodilators such as histamine sympathetheticolytic and adrenolytic compounds such as tetræthylammonium chloride and benzazoline hydrochloride, anticoagulants, circulatory stimulants such as ephedrine, caffeine and deoxycortone acetate, vitamins including $B_{12}$ and various miscellaneous agents such as trypan red and vaccines For symptomatic relief of spasticity drugs have been given such as quinine neostigmine Tridione, curare and mephensin

General measures include the avoidance of fatigue and the use of hot baths to diminish spasticity

### Neuromyelitis Optica

*(Devic s Disease)*

This is another demyelinating disease characterised by disseminated myelitis retrobulbar neuritis and at times papillœdema Clinically there is blindness with paraplegia, sensory loss and incontinence In some cases there is almost complete recovery, but the death rate is about 50%

### Syringomyelia

*(Syringoencephalomyelia)*

**Definition** A disease characterised by a peculiar sensory disturbance muscular wasting and trophic lesions due to cavity formation and neuroglial overgrowth in the cord and mid brain

**Etiology** The cavity formation is generally believed to be due to a developmental defect the central canal of the cord not closing properly It is possible that in some cases there is first an overgrowth of neuroglial tissue, which breaks down to form a cavity *Predisposing causes* 1 Age Usually the symptoms are first noted between 10 and 60 years frequently between 25 and 40 2 Sex The incidence is rather greater in the male Trauma to the spine may be a predisposing cause in some cases

**Pathology** The spinal cord shows characteristic changes The dura is usually normal but the pia may be thickened The cavity is generally situated posterior to the central canal in the posterior commissure It is most likely to be present in the lower cervical and upper thoracic regions, running up and down the cord for a variable distance Thus it may extend for a few segments or run the length of the cord and into the medulla The cavity may branch and vary in size from a small hole to one occupying the greater part of the transverse section of the cord The cord may be distended and cause pressure on the vertebræ The cavity may contain a thin watery or glairy fluid and may communicate with the central canal of the cord and be lined with ependymal cells The neuroglial overgrowth occurs around the cavity, and isolated masses of neuroglial tissue may be present at varying sites in the cord The cavity and the surrounding neuroglial overgrowth are most likely to interfere with the nerve fibres entering

**Etiology** The disease is usually associated with pernicious anæmia less often with other types of anæmia In some cases the anæmia is slight whereas the nervous changes are severe *Predisposing causes* 1 Age 30 to 65 years 2 Sex The incidence is equal In some cases there is a familial occurrence of the disease

**Pathology** The lower thoracic part of the cord is usually the first site of the lesion There is degeneration of the white matter which begins in the posterior or lateral columns in irregular patches These tend to fuse and to spread up and down the cord in a funicular manner and the internal capsule of the brain may be affected The medullary nerve sheaths and later the axons in the cord degenerate There is very little tendency to a proliferation of neuroglial tissue The peripheral nerves may show degeneration but the grey matter of the cord and the nerve roots themselves are not usually affected Changes characteristic of pernicious anæmia may be found in other parts of the body

**Clinical Findings** The patient is usually an adult of middle age who complains of the gradual onset of peculiar feelings in the legs or arms such as numbness pins and needles burning or pains These sensations are frequently first felt in the toes or fingers and later extend up the limbs Sharp lightning pains may also be felt in the limbs or a girdle' sensation around the trunk The legs gradually feel heavy and walking is an effort When the eyes are closed or when the patient is in the dark he may feel definitely unsteady Impotence is the first symptom in some cases Often the blood shows evidence of pernicious anæmia when the nervous symptoms appear although it is rare for subacute combined degeneration to develop in a patient who has been adequately treated for pernicious anæmia Many cases have however been recorded in which no definite anæmia was detectable when the nervous symptoms appeared An acute onset is more rare with fever vomiting and diarrhœa and pains in the back and legs

*On Examination* Spinal sensory system Changes are usually first detected here Anæsthesia for pain temperature and touch may be found in the periphery of the legs or arms in a stocking or glove area Vibration sense disappears in the tibiæ The joint sense of the big toe is lost The calf muscles may be unduly tender on pressure On the trunk segmental areas of anæsthesia may be detected Spinal motor system The muscles of the legs become weak and their tone increased but in some cases there is a flaccid paresis There may be wasting of the small muscles of the hands The reflexes The knee jerks and ankle jerks are usually exaggerated and an extensor plantar response is obtained In the flaccid type of paresis the knee jerks and ankle jerks are sluggish but the plantar response here is usually extensor The abdominal reflexes are generally exaggerated The sphincters become paralysed late in the disease Co-ordination Loss of postural sense gives rise to inco ordination and Rombergism The gait This is ataxic Trophic changes Œdema of the subcutaneous tissues and bed sores are liable to develop in advanced cases The cranial nerves In some instances there is primary optic atrophy or optic neuritis

*The bulbar type (syringobulbia)* Bulbar symptoms may occur in the *cervical type, or independently* The lesions are usually unilateral such as laryngeal paralysis dysphagia, wasting of half of the tongue, nystagmus and ocular paresis, anæsthesia of half of the face, or facial weakness Further, certain clinical types are described such as 1 The classical type depicted above under the cervical variety 2 The motor type in which motor symptoms predominate and resemble those of amyotrophic lateral sclerosis 3 The sensory type which may be *mistaken for hysteria* 4 The trophic type, here all sensation may be lost in the hands and painless whitlows and ulcers occur (Morvan s disease) 5 The tabetic type, in which the lesion spreads into the posterior columns, and there are loss of knee jerks Rombergism severe pains and arthropathies of the legs

**Differential Diagnosis** The diagnosis of syringomyelia is established by the presence of dissociated anæsthesia Difficulty occurs in cases *in which motor symptoms predominate or are first noted* Then other conditions such as a cervical rib progressive muscular atrophy amyotrophic lateral sclerosis disseminated sclerosis and lateral sclerosis may require exclusion Horner s syndrome may also result from thrombosis of the posterior inferior cerebellar artery a cervical rib a spinal cord tumour disseminated sclerosis or a hæmorrhage at the level of C 7 to Th 1 cervical pachymeningitis an aortic aneurysm, mediastinal or cervical tumour, apical lung carcinoma, apicolysis thoracoplasty, trauma, stellate ganglionectomy or occur in partial facial hemiatrophy (the Parry Romberg syndrome) In hæmatomyelia the signs may closely resemble those of syringomyelia but in the former the onset is more sudden and as the blood is absorbed the signs gradually disappear

**Course and Complications** Syringomyelia usually pursues a slowly progressive course lasting from 10 to 20 years or longer but for a considerable time the symptoms and signs may remain stationary Hæmorrhage may take place into the cavity causing pain or paraplegia Other complications include cystitis, pyelonephritis and pulmonary tuberculosis

**Prognosis** This is unfavourable Death is usually imminent if there are extensive mid brain lesions

**Treatment** No cure is known Care should be taken to protect anæsthetic areas from injury Deep X ray treatment to the affected area of the medulla or spinal cord may relieve pain improve the trophic changes and increase muscular power but in many cases it is not successful Pains may also be relieved by tab codein co 1 t i d Operations to drain the cavity or relieve pressure on the vertebræ are often fatal

## Subacute Combined Degeneration of the Cord

### *(Subacute Combined Sclerosis)*

**Definition** A disease characterised by degeneration of the posterior and lateral columns of the cord with anæmia of the pernicious type and achylia gastrica

5 millions per c mm Folic acid should not be given as it may actually lead to neurological changes

## Friedreich's Ataxia

*(Hereditary Spinal Ataxia)*

**Definition** A disease characterised by ataxia due to sclerosis of the posterior spino cerebellar and pyramidal tracts of the cord There is a familial or hereditary tendency

**Etiology** The cause is unknown but it may be due to a premature degeneration (abiotrophy) of certain nerve fibres *Predisposing causes* 1 Heredity The disease tends to occur in several members of a family and at times in several generations of the same family 2 Age Usually between 5 and 15 years rarely it does not develop until about the age of 30 3 Sex Both sexes are equally affected

**Pathology** The spinal cord may appear smaller than normal There is atrophy of the nerve fibres of the following tracts The posterior columns the spino cerebellar tracts of Gowers and Flechsig and the direct and crossed pyramidal tracts Clarke s column is also affected Sclerosis results from a secondary overgrowth of neuroglial tissue The changes are most marked in the lumbo sacral part of the cord The cerebellum is usually normal although some cellular degeneration may occur In some cases congenital pulmonary stenosis is also present

**Clinical Findings** The onset is insidious The mother may notice a gradually developing deformity of her child s feet or that there is difficulty and clumsiness in walking The latter may be intensified in the dark

*On Examination* The nervous disease case sheet described on p 298 can be filled in as follows —

## Case Sheet

*(Friedreich s Ataxia)*

*Age* 5 to 15 *Sex* Male or female

*Cerebration* The child may be backward *Speech* Slurred

*Cranial Nerves* I Normal II Optic atrophy may occur III IV and VI Lateral nystagmus may be present V (a) Motor Tremors of masticatory muscles may occur (b) Sensory Normal VII (a) Motor Twitching of the facial muscles may occur (b) Sensory Normal VIII Normal IX X and XI Usually normal XII Tremors of tongue may occur

*Spinal Motor Nerves* (a) Power There is weakness of the legs and perhaps of the arms (b) Wasting This may occur late in the weak muscles (c) Tone The affected muscles are usually flaccid (d) Contractions Contractures of the foot muscles result in pes cavus hyperextension of the big toe and hammer toes Kyphoscoliosis is also due to muscular action Tremors of the head neck and trunk are often seen and intention tremors of the hands and arms so that there is difficulty in picking up fine objects

*Spinal Sensory Nerves* (a) Cutaneous sensation This is usually

Nystagmus may also be present. Involvement of the cervical sympathetic will give rise to enophthalmos and constriction of the pupil usually on one side. *Mentality.* There may be a gradual lowering of the mental calibre of the patient. *The cerebro spinal fluid.* This is normal. *Electrical reactions.* There is usually no reaction of degeneration, but a gradual diminution of response to electrical stimuli occurs. *General examination.* The tongue is often smooth and shiny. *The blood.* Changes typical of pernicious anæmia are generally found in cases of some duration. The fractional test meal shows achylia gastrica, as in pernicious anæmia. The spleen may be palpable. During the course of the disease an irregular degree of fever may be noted.

**Differential Diagnosis.** In the early stages the symptoms and signs may be those of peripheral neuritis and the weakness of the legs and stocking or glove anæsthesia may lead to a diagnosis of hysteria. The occurrence of an extensor plantar response however puts this diagnosis out of court. When the posterior columns are chiefly affected the case resembles one of tabes dorsalis but this again is excluded by an extensor plantar response the achylia, and the normal cerebro spinal fluid. Involvement of the lateral columns of the cord causes the disease to resemble disseminated sclerosis, Friedreich s ataxia amyotrophic lateral sclerosis, primary lateral sclerosis or a cord tumour. The combination of evidence of a posterior and lateral column cord lesion with achylia gastrica and a blood count resembling that of pernicious *anæmia, enables the diagnosis of subacute combined degeneration to* be made but in some cases no significant changes are found either in the blood or the bone marrow although a histamine fast achlorhydria is almost always present.

**Course and Complications.** The disease usually pursues a gradually progressive course. In some cases it may be divided into three stages first that of ataxic paraplegia, this is followed by spastic paraplegia with marked evidence of anæsthesia and finally there is a flaccid paraplegia with loss of sphincter control. It is comparatively rare for the disease to pursue a rapid course of a few weeks or months. Complications include cystitis, pyelitis pyelonephritis and bed sores.

**Prognosis.** Before the introduction of liver treatment death occurred in 2 to 3 years from the onset of symptoms. The outlook however has been completely altered by liver treatment as cases have now been recorded in which the patient has been enabled to return to work and *such signs of organic nervous lesions as the extensor plantar response and* sensory loss have disappeared. The blood count also returns to normal.

**Treatment.** The patient should be given 5 ml of a liver extract such as Neo Hepatex or Hepastab intramuscularly daily until he can walk, and subsequently a maintenance dose of 10 ml must be injected every 1, 2, 3 or 4 weeks. Alternatively vitamin $B_{12}$ can be injected in doses of 100 micrograms weekly for 6 months. The dose may then be reduced to 50 micrograms a week. Later 50 to 150 micrograms a month may suffice. In all cases the red cells should be maintained at

*Brown s ataxia* This also resembles Friedreich s ataxia The chief distinguishing points of Sanger Brown s ataxia are Optic atrophy is generally present there is no nystagmus and the plantar reflexes are flexor It also usually develops at a later date than does Friedreich s ataxia

## Peroneal Muscular Atrophy

(*Charcot Marie Tooth Type of Muscular Atrophy Progressive Neural Muscular Atrophy*)

**Definition** A disease characterised by wasting of the small muscles of the feet and hands and the muscles of the distal parts of the extremities associated with lesions in the central nervous system

**Etiology** The cause is unknown *Predisposing causes* 1 There is a familial tendency and it appears to be transmitted by females 2 Age Between 5 and 10 years or in early adult life 3 Sex Males predominate slightly

**Pathology** Degeneration of the anterior horn cells occurs in the sacral lower cervical and upper thoracic regions of the cord Degeneration may also be present in the cells of Clarke s column and in the posterior and postero lateral columns of the cord The motor nerves supplying the affected muscles atrophy there is usually an interstitial neuritis of the branches of the external popliteal nerve and atrophic or fibrotic changes occur in the wasted muscles

**Clinical Findings** The disease starts gradually the patient experiencing difficulty in walking as the feet tend to drop and become inverted A condition of bilateral talipes equino varus thus develops Later as the muscles in the legs are affected there is greater difficulty in walking After a few years wasting is noticed in the intrinsic muscles of the hands with tremors and the muscles of the forearms are subsequently affected

*On Examination* During the active stages of the disease fibrillary twitchings are seen in the muscles which are wasting Contractures tend to occur so that the feet are clubbed and the hands clawed The thighs are not affected except that in some cases there may be muscle wasting just above the knees the upper arm trunk neck and face muscles are unaffected The limbs assume the shape of a bottle with the neck downwards The knee jerks are brisk but the ankle jerks are lost The superficial reflexes are present but the plantar response is sluggish or difficult to obtain owing to the contractures Sensation may be normal or there may be varying degrees of loss of cutaneous sensation over the lower parts of the arms and legs Vibration muscle and joint sense may also be abolished A reaction of degeneration is usually present in the affected muscles

**Differential Diagnosis** The disease cannot be distinguished with certainty in the early stages from progressive muscular atrophy which may start in the feet Later however the characteristic distribution of the muscular atrophy establishes the diagnosis In peripheral neuritis the onset is more acute and the sensory changes are more

normal It may be slightly blunted on the feet (*b*) Joint sense This is often normal but may be lost later in the big toe (*c*) Kinæsthetic sense Often lost later (*d*) Muscle and tendon sense This may be normal or lost in the legs (*e*) Nerve sensation This is usually normal (*f*) Stereognostic sense Usually normal (*g*) Vibration sense This may be lost over the tibiæ (*h*) Subjective There are usually no pains

*The Reflexes* (*a*) Superficial 1 Conjunctival and corneal *Normal* 2 *Palatal Normal* 3 *Pharyngeal Normal* 4 5 and 6 Epigastric, abdominal and cremasteric are lost late in the disease 7 Plantar Extensor (*b*) Deep 1 Pupil Reaction sluggish to light and accommodation 2 Jaw jerk *Not present* 3 4 and 5 Biceps, triceps and supinator jerks may be absent 6 and 7 Knee jerk and ankle jerk Lost (*c*) Visceral Normal

*Co ordination* There is often inco-ordination of the arms and legs, but Rombergism may not be present

*Trophic Changes* Bed sores may occur in the terminal stages *Gait* Reeling The patient stands on a wide base Static ataxia is often seen, the patient swaying when he is standing still *Electrical reactions* Normal *Lumbar puncture* Fluid normal

**Differential Diagnosis** Juvenile tabes is excluded by the familial incidence foot deformity extensor plantar response and absence of pupillary changes in Friedreich s disease In disseminated sclerosis the knee jerks are exaggerated, and the age incidence is usually later In Marie s hereditary ataxia the onset again is later the knee jerks are exaggerated and there are no deformities of the feet

**Course and Complications** The course is usually slowly progressive the patient eventually being confined to bed

**Prognosis** In some cases the disease is arrested, and frequently the patient may live for over 30 years from the onset

***Treatment*** *There is no known cure The limbs should be massaged* and walking exercises encouraged Special boots are usually required

## Spino cerebellar Ataxia

### (*Marie s Hereditary Cerebellar Ataxia*)

This disease resembles in many respects Friedreich s ataxia The onset is usually later generally after the age of 20 years It shows both a hereditary and a familial incidence There may be some degeneration of cells in the cerebellum but the chief lesion is in the spino cerebellar tracts of the cord especially in the dorsal cerebellar tract of Flechsig The gait is of a reeling character there is inco-ordination of the legs and later of the arms The speech is slurred and optic atrophy and nystagmus may be present *The knee jerks* are usually exaggerated The disease differs from Friedreich s ataxia in several points such as the absence of deformity of the feet or of kyphoscoliosis the presence of muscular hypertonia and the exaggerated knee jerks Optic atrophy is more common than it is in Friedreich s ataxia Another type of spino cerebellar ataxia is known as *Sanger*

## THE SPINAL NERVES

### Neuralgia of the Spinal Nerves

**Definition** Pain along the course of a nerve not associated with a detectable organic lesion

**Etiology** There is little distinction between neuralgia and slight degrees of neuritis and both are probably due to similar causes These include inflammation of the nerve sheath or sensory ganglion anæmia cachexia and toxic substances such as alcohol and lead Bacterial and protozoal infections may also be associated with neuralgia as in influenza syphilis and malaria Exposure to cold may also produce an attack *Predisposing causes* 1 Age Usually adults 2 Sex Females predominate

**Clinical Findings** The patient complains of pain in the course of a spinal nerve It often occurs in paroxysms and may tend to recur daily at about the same time

*On Examination* Tenderness may be found at certain points on the course of a nerve or the skin may be hyperæsthetic over the area supplied by the nerve Certain varieties will be mentioned *Cervico occipital neuralgia* Pain occurs in the region of the great occipital nerve over the back of the head The skin may be very sensitive to the touch *Brachial neuralgia* The pain is situated in such sites as the outer part of the shoulder in the area of the circumflex nerve in the upper arm or forearm or in the region of the median or ulnar nerves *Intercostal neuralgia* The pain is located to an intercostal space and tender spots may be found at the sites of emergence of the lateral or anterior cutaneous branches (see Fig 22A) *Crural neuralgia* The pain occurs along the front and inner aspect of the thigh *Sciatic neuralgia* Here the pain is felt in the region of the great sciatic nerve or its terminal divisions *Coccydynia* The pain is related to the coccygeal plexus and is felt in the region of the coccyx *Plantar neuralgia* This may be associated with gonorrhœa

**Differential Diagnosis** Every endeavour should be made to exclude a true neuritis especially that due to pressure by a tumour or enlarged glands There are no muscular signs and usually no loss of sensation in neuralgia

**Course and Complications** The course is often prolonged and there is a tendency to recurrence

**Treatment** The patient should be thoroughly examined to exclude any general cause of ill health such as anæmia or chronic infection Local treatment in the form of heat is of great value This may be applied by hot bottles or by diathermy A sedative mixture con taining Tnc gelsemii m 10 sod bicarb gr 15 sod salicyl gr 5 sod brom gr 10 syr aurant m 20 aquam ad fl oz ½ Fl oz ½ ex aqua t d s will often afford relief Other sedatives such as aspirin gr 10 to 15 may be used to afford relief or to secure sleep Opium preparations should not be prescribed for fear of establishing a habit

marked In the distal type of myopathy of Gowers and Spiller (see p 625) there are no sensory changes and fibrillation is not present Claw hand and talipes equino varus also occur in progressive hyper trophic neuritis (Dejerine Sotta's disease) but here acute pains occur in the arms and legs, and the peripheral nerves are thickened and palpable

**Course and Complications** The course is usually slowly progressive over several years, but arrest of the disease then occurs as shown by the absence of fibrillary twitching

**Prognosis** The disease is not fatal but the affected muscles do not recover although an improvement of function often occurs due to the formation of fibrous tissue

**Treatment** The muscles should be massaged Operations are contra indicated but light splints should be worn to help to prevent and correct deformities

## Progressive Spinal Muscular Atrophy of Infants

*(Werdnig Hoffmann Disease)*

**Definition** A disease of infants characterised by progressive weakness of muscles due to degeneration of their motor neurones

**Etiology** The cause is unknown *Predisposing causes* 1 The disease may occur in several members of a family 2 Age The onset is usually shortly after birth 3 Sex Both sexes are affected

**Pathology** There is degeneration of the anterior horn cells in the cord supplying the muscles involved with subsequent degeneration of the anterior nerve roots There may also be degenerative changes in the bulbar nuclei in the posterior columns and the pyramidal tracts The affected muscles are atrophied

**Clinical Findings** The weakness of the muscles is usually noticed within 2 months of birth or it may not show itself until towards the end of the first year The muscles are flabby but the nutrition of the infant is generally good The muscles first affected are those of the trunk and pelvic girdle the limb muscles proximal to the trunk are subsequently involved and later the neck muscles in a terminal stage the muscles supplied by the bulbar nuclei may atrophy The infant lacks power to support itself and move as a normal child and if it survives cannot walk at the normal age Fibrillary twitchings are not usually seen in the affected muscles There is loss of both superficial and deep reflexes Sensory changes are sometimes detected

**Differential Diagnosis** When the disease is present at birth it closely resembles amyotonia congenita (see p 626) In the latter however, there is a tendency towards recovery

**Course and Complications** The disease is usually rapidly progressive Pulmonary complications such as bronchitis or broncho pneumonia may develop

**Prognosis** The disease is fatal in the course of a few weeks or months

**Treatment** There is no known treatment

4 or 5 days followed by subcutaneous injections of vitamin $B_{12}$ 100 micrograms weekly for 6 weeks An alternative treatment is the intramuscular injection of 5 ml of gamma globulin into each gluteal region every second day until the vesicles begin to disappear Pain is relieved by such drugs as aspirin gr 10 t d s or tab codein co 1 t d s For post herpetic neuralgia treatment with X rays to the affected region of the spine should be tried and a sedative such as pheno barbitone gr ½ to 1 taken at night In very intractable cases chordo tomy or alternatively division of the posterior nerve root between the affected ganglion and the cord together with the roots immediately above and below may be advised In some cases however these operations fail to relieve the pain

## Radiculitis

**Definition** Inflammation or degeneration of the spinal nerve roots

**Etiology** Radiculitis may be due to intervertebral disc disease to disease of the vertebræ such as caries or spondylitis to meningitis due to syphilis or tuberculosis to trauma usually produced by traction on a limb or to the pressure of a cervical rib

**Clinical Findings** The patient complains of pain often burning in character which may be paroxysmal It is usually located to a band area around the trunk or along an arm or leg

*On Examination* Hyperæsthesia is generally present in the early stages over the site of the pain In long standing cases the painful area may become anæsthetic Bone vibration sense may also be lost in the affected part Muscular wasting occurs in the groups of muscles supplied by the affected nerve roots The cerebro spinal fluid may show an excess of lymphocytes

**Treatment** This varies with the cause and is described under the appropriate sections

## Neuritis

**Definition** Inflammation or degeneration of a nerve

**Etiology** Localised neuritis may be due to various causes such as 1 Trauma 2 Compression by a tumour or enlarged glands 3 Fibrositis involving the nerve sheath 4 Toxins derived from micro organisms and possibly from the intestine 5 Division of the nerve results in degeneration Cold is a predisposing cause Multiple neuritis is separately considered (see p 444)

**Pathology** In interstitial neuritis inflammatory changes occur in the connective tissue between the nerve fibres In pressure or section neuritis there is degeneration of the nerve sheath and axis cylinders

**Clinical Findings** The clinical findings vary in different cases according to which function of the peripheral nerve is chiefly affected and also according to the nature of the lesion Thus the symptoms may be chiefly motor sensory or trophic but these are usually com bined If the nerve is irritated as in fibrositic infiltration, the chief clinical feature is pain of a varying degree Pain of a burning character is known as causalgia The muscles supplied by the

## Herpes Zoster

(*Shingles Zona The Radiculo ganglionic Syndrome*)

**Definition** A painful condition associated with a cu
eruption and inflammation of the posterior nerve root ganglia

**Etiology** The cause is probably a virus The so-called symp
zosters are examples of the virus infection being associated with
diseases such as cerebro spinal meningitis, myelitis, subara
hæmorrhage, spinal carcinoma or treatment with bismuth, ars
gold In some cases herpes zoster is associated with varicella th
disease occurring in a person who is in contact with a patient su
from herpes *The virus of herpes may be a mutant of the chick*
virus It may also occur in epidemics An attack of varicella d
afford protection against herpes

**Pathology** There is inflammation of one or more posteri
ganglia The ganglion swells and there is lymphocytic infilt
Acidophil nuclear inclusion bodies are found in the epithelial
the vesicles

**Clinical Findings** The patient first complains of pain or of
algesia in the region of a spinal nerve usually in the chest or trun
at times along a limb In the course of 1 or 2 days the cut
eruption appears This consists of vesicles containing clear fluid o
background The temperature is sometimes raised to 99° or 100°
a few days at the onset The cerebro spinal fluid may show an ex
globulin and of lymphocytes Certain clinical varieties are desc
such as zoster universalis (which may be confused with var
ophthalmic zoster geniculate zoster and glossopharyngeal and
zosters Zoster encephalitis and myelitis have been recorded I
*latter the pyramidal tracts may be involved*

**Differential Diagnosis** In the early stages the pain may be mis
for that of pleurisy or neuritis The appearance of the er
establishes the diagnosis Herpes febrilis, which occurs in such
tions as the common cold and lobar pneumonia does not follow a
distribution and attacks are liable to recur The causative viru
differs from that of herpes zoster and it produces encephalitis
*injected into rabbits*

**Course and Complications** The vesicles dry up in about a
leaving permanent scars Severe neuralgia may occur
complication Lower motor neurone paresis involving the affect
adjacent segments is an occasional complication Otic herpes ma
accompanied by facial palsy or ophthalmic herpes by oculomotor w
ness, chemosis iritis or cyclitis Immunity is usually conferre
one attack

**Prognosis** This is good but in elderly people the post her
neuralgia may be very intractable causing insomnia

**Treatment** The skin lesion should be kept dry a powder of
and zinc oxide being applied or as is often more convenient a g

obliterans and polyarteritis nodosa 6 Acute infective or toxic polyneuritis is thought to be caused by an unknown toxin or virus *Predisposing causes* 1 Age Usually 20 to 40 but children are affected in diphtheria 2 Sex Equal incidence 3 Cold and exposure may predispose In some cases no cause is discovered

**Pathology** In long standing cases there is parenchymatous degeneration of the peripheral nerves usually in their distal parts The toxin is probably carried by the blood stream and appears to have a special affinity for certain nerves and in some cases for certain nerve fibres in the nerves Thus in lead poisoning the motor fibres supplying certain arm muscles are especially liable to be affected Chromatolytic changes may also be seen in the cerebral cortical cells

**Clinical Findings** The onset is usually insidious with prodromal symptoms Thus the patient may complain of numbness tingling or pains in the legs or of cramps in the calves The feet or legs may feel hot or cold Various clinical types of neuritis exist according to the toxic agent Thus in lead neuritis the motor nerve fibres are affected usually in the arms but sometimes in the legs or elsewhere Both arms or legs are involved the distribution of the lesion being symmetrical The motor lesions are of the lower neurone type there being a flaccid paralysis of certain muscles with wasting and loss of the deep reflexes A reaction of degeneration may be obtained Often a mixed type of neuritis is present with motor sensory and possibly trophic changes Thus in alcoholic neuritis the legs are chiefly affected There is pain in the calves or feet with muscular tenderness Sensory changes may be present with a stocking area of distribution Hyperæsthesia or anæsthesia of the foot and lower part of the legs may be found In the early stages the knee jerks may be exaggerated but later they are lost In diabetic neuritis trophic changes may be seen such as a perforating ulcer or gangrene of a toe Other trophic changes found in different types of neuritis include vasomotor disturbances such as redness of the skin a smooth glossy skin absence of hair increased sweating œdema brittleness of the nails and keratodermia Achlorhydria is present in some cases of multiple neuritis

In acute infective polyneuritis there is a rapidly spreading paralysis involving the legs arms and trunk The VII cranial nerve may also be affected There is in addition constitutional disturbance as shown by the rise of temperature *The Guillain Barre syndrome* is characterised by a diffuse polyneuritis often with bilateral facial palsy Bulbar paralytic symptoms may develop The cerebro spinal fluid contains an excess of albumin but no cells Fever is usually absent except at the onset The mortality rate varies between 8% and 30%

Differential Diagnosis In the motor variety of peripheral neuritis other causes of weakness and wasting of muscles must be considered The symmetrical distribution of a lower motor neurone lesion is characteristic of peripheral neuritis In anterior poliomyelitis the illness is acute and special groups of muscles are picked out The temperature is also usually raised In acute myelitis the paralysis involves the lower part of the body to a certain level the sphincters are usually

nerve are tender, and often cramps, or painful muscular contractions occur when the limb is at rest The deep reflexes are often exaggerated When there is more definite compression neuritis there is muscular wasting of an atrophic type with weakness The muscles are also tender and there is alteration of cutaneous sensibility Pain and temperature sensations are abolished over the central part of the affected area but around this there is a zone in which touch sensation is absent but pain is felt more acutely than normal There is usually no reaction of degeneration If the nerve is completely severed the muscles supplied are paralysed, flaccid and wasted and the reaction of degenera tion is present The deep reflexes are lost The muscles are not tender, and there is complete loss of cutaneous sensation Trophic changes include dryness and smoothness of the skin a localised sweating and keratosis may occur There may also be redness or blueness or ulceration of the skin *In brachial neuritis* pain, numbness or tingling may be felt above the clavicle, in the axilla arm or hand *In ulnar neuritis* pain occurs in the arm or forearm on the inner side The nerve is tender on pressure at the elbow Sensation may be blunted over the inner part of the hand and the inner one and a half fingers *In median neuritis* a severe burning pain (causalgia) may be felt in the palm of the hand with alteration of sensation in the outer part of the hand and outer three and a half digits

**Treatment** During the acute stage the patient should rest in bed the arm being supported on a pillow A liniment of 1 part of lin aconit, 2 parts of lin belladon and 3 parts of lin chlorof (B P C) should be applied daily to the arm, which is then covered with cotton wool Electrical treatment is best avoided during the acute stages Sleep may be secured by the use of hypnotics, such as Medinal gr 5 to 10 and aspirin gr 10 t i d or tab codein co 1 t i d taken for relief of pain After the acute stage has subsided light massage should be applied to the arm and if adhesions have formed in the shoulder joint, they will probably disappear as the arm is used A search for a septic focus in the mouth nasopharynx alimentary or renal tracts should also be made and if found eliminated

Meralgia paræsthetica is described on p 455

## Multiple Neuritis

(*Polyneuritis Peripheral Neuritis*)

**Definition** Inflammation or degeneration of several peripheral nerves

**Etiology** The causes which are varied may be grouped as follows 1 Exogenous chemical substances such as alcohol arsenic lead mercury copper phosphorus silver gold ether, carbon monoxide bar bitone, etc 2 Toxins of micro organisms as in diphtheria enteric fever influenza gonorrhœa syphilis malaria, leprosy and tuberculosis 3 Deficiency diseases such as beri beri or lack of the vitamin B complex 4 Metabolic disturbances as in diabetes mellitus, gout anæmia, porphy ria, and carcinoma 5 Vascular causes such as thrombo angitis

situated under the skin they are usually tender to pressure (tuberculosa dolorosa)

**Treatment** If the tumour is causing symptoms and is accessible it should be removed surgically

## The Cervical Plexus and its Branches

**Anatomy** The cervical plexus is formed from the anterior primary divisions of the first four cervical nerves

### The Phrenic Nerve

**Anatomy** The phrenic nerve is formed from C 3 C 4 and C 5 nerves chiefly from C 4 It passes down the neck over the scalenus anticus muscle and after traversing the thorax supplies the diaphragm.

**Etiology of Lesions** Lesions may be due to 1 Trauma such as a wound in the neck. 2 Pressure as by arthritis or fracture of the spine enlarged glands a mediastinal tumour or abscess or a pericardial effusion 3 Nervous diseases such as spinal meningitis poliomyelitis hæmorrhage of the cord Landry s paralysis neuritis or a tumour 4 Avulsion or crushing as in the treatment of pulmonary tuberculosis

**Clinical Findings** Observation in cases of avulsion of the phrenic nerve shows that usually no abnormal physical signs can be detected Thus the movement of the lower part of the chest and of the upper part of the abdomen appears normal The air entry at the base of the lung on the affected side is often weaker owing to the fibrosis of the lung present in such cases X ray examination shows the diaphragm on the affected side raised 1 or 2 inches and motionless or exhibiting paradoxical movement (see p 210) In cases of paralysis of the diaphragm by enlarged glands due to malignant disease reversal of diaphragmatic movement may occur on the affected side the paradoxical movement showing the affected half of the diaphragm to ascend with inspiration when the diaphragm on the sound side descends Spasm and flutter of the diaphragm are considered on pp 210 212 If both phrenic nerves are paralysed there is recession of the upper part of the abdomen with inspiration If the intercostal muscles are also paralysed death occurs from respiratory failure

## The Brachial Plexus and its Branches

**Anatomy** The brachial plexus is formed from the anterior primary divisions of the 5th to 8th cervical nerves and from part of the 1st thoracic nerve

**Etiology of Lesions** The brachial plexus may be affected by various lesions such as 1 Lesions of the cervical spine such as the *Klippel Feil syndrome* in which there is fusion of the vertebræ prolapsed inter vertebral disc cervical spondylosis fracture dislocation and caries due to tuberculosis syphilis or secondary deposits 2 Trauma from a wound or by violent traction of the arm as occurs in obstetrical paralysis 3 Pressure from a cervical rib a rudimentary first rib or a normal first rib or from a fractured or dislocated clavicle or humerus or a subclavian aneurysm 4 Neuritis (see p 443)

affected, which is not the case in peripheral neuritis and sensory changes extend up to a definite segmental level Landry's paralysis closely resembles acute infective polyneuritis When ataxia and pains in the limbs are prominent features tabes dorsalis may be suggested In the latter the anaesthetic patches on the trunk and nose the pupil changes and the examination of the cerebro spinal fluid are diagnostic The trophic type may suggest Raynaud s disease erythromelalgia acro cyanosis or syringomyelia There are usually some motor changes present as well in peripheral neuritis and the stocking and glove distribution of sensory changes is suggestive of peripheral neuritis

**Course and Complications** The course is usually chronic the symptoms lasting several months In acute infective polyneuritis the course is however, more rapid Mental changes, such as Korsakow's psychosis (see p 778) are especially liable to occur in association with alcoholic neuritis Myocardial degeneration may also be present, particularly in diphtheritic neuritis and in beri beri Permanent contracture of the antagonistic muscles may ensue

**Prognosis** This is improved when the diagnosis is made early and the cause removed at once In acute infective polyneuritis the outlook is serious and death may rapidly result from respiratory or cardiac failure

**Treatment** The patient should be put to bed and the affected limbs rested If there is evidence of myocardial degeneration the patient should lie with the head raised on one pillow only The cause of the neuritis should if possible be removed Contracture of the unaffected muscles must be prevented by the use of light celluloid splints to keep the affected muscles from being over stretched Hot fomentations are useful in relieving pain In addition it may be necessary to give analgesic drugs such as aspirin gr 10 or tab codein co 1 t d s Special treatment is required for the neuritis due to lead (see p 775) and diphtheria (see p 567) In the Guillain Barré syndrome favourable results have followed the daily intravenous saline drip injection of mg 25 ACTH Treatment with vitamin $B_1$ or with the whole vitamin B complex is of doubtful value in alcoholic neuritis Electrical treatment by diathermy is of value in some cases for the relief of pain As the pain subsides the muscles should be gently massaged

## Tumours of Nerves

**Etiology** The cause is unknown

**Pathology** The tumour may be a neuroma a fibroma or sarcoma Single or multiple tumours may be met with The true neuroma consists of nerve ganglion cells or nerve fibres and occurs in the sympathetic system The perineurial fibroblastoma arises from the nerve sheath When appearing on cutaneous nerves they are known as molluscum fibrosum and if in addition there is pigmentation of the skin they constitute von Recklinghausen s disease

**Clinical Findings** Nerve tumours may give rise to no symptoms If attached to the posterior nerve roots they may cause pain or muscular weakness if they arise from the anterior nerve roots When

and the brachial plexus by division of the first rib For acroparæsthesia, the only treatment that affords relief is rest electrical treatments are useless

### The Long Thoracic Nerve

*(The Posterior Thoracic Nerve The Nerve of Bell)*

**Anatomy** The long thoracic nerve arises from the C 5 C 6 and C 7 nerve roots as they traverse the intervertebral foramina It passes through the scalenus medius muscle to the axilla and to the outer side of the serratus magnus muscle which it innervates

**Etiology of Lesions** The long thoracic nerve may be affected by 1 Trauma by a wound in the neck or axilla or by carrying heavy weights on the shoulder as may occur with porters 2 Neuritis in influenza or diphtheria 3 A cord or cerebral lesion in progressive muscular atrophy

**Clinical Findings** The patient finds that his upper arm and shoulder are weak Thus there is difficulty in elevating the arm above the level of the horizontal and in pushing with the arm

*On Examination* When the arm is by the side the scapula on the affected side is higher than the other one and the inferior angle is nearer the spine If the patient is asked to hold his arms out in front of him and the back is inspected there is prominence of the vertebral border of the scapula (winging of the scapula) on the affected side

### The Circumflex Nerve

**Anatomy** The circumflex nerve arises from the posterior cord of the brachial plexus from the C 5 and C 6 nerves It travels behind the axillary artery and winds round the surgical neck of the humerus to supply the deltoid and teres minor muscles It also gives a branch to the shoulder joint and a cutaneous nerve to the skin of the upper and outer half of the arm

**Etiology of Lesions** The circumflex nerve may be affected by 1 Trauma in dislocation or fracture of the shoulder or by the pressure of a crutch 2 Arthritis and bursitis of the shoulder 3 Neuritis in fibrositis diabetes mellitus or exposure to cold

**Clinical Findings** The patient complains of pain in the region of the shoulder and the outer and upper part of the arm He may also experience difficulty in raising the arm or rotating it outwards

*On Examination* Flattening of the shoulder may be seen owing to wasting of the deltoid There is some blunting of sensation over the upper and outer part of the arm The pain may encircle the upper part of the arm like a hot wire completely preventing sleep and it is always worse when the patient is in bed In long standing cases adhesions may form in the shoulder joint

### The Musculo spiral Nerve

**Anatomy** The musculo spiral nerve is formed from the posterior divisions of C 5 C 6 C 7 and C 8 nerve roots

**Clinical Findings** If the lesion affects the whole of the brachial plexus, the corresponding arm and hand *are flaccid and paralysed*, and sensation is lost over the arm and hand The sympathetic supply to the eye may also be involved by the injury to the Th 1 root and the eye on the same side *shows a small pupil with enophthalmos* and possibly slight ptosis Only a portion of the plexus may be damaged Thus if the C 5 and part of the C 6 roots are injured there is paralysis *of the upper arm type (Erb Duchenne) This is the* type met with in obstetrical paralysis The muscles paralysed include the deltoid biceps, brachialis anticus, and the supinator longus but the *sensory loss is usually slight If the C 8 and Th 1 roots are* injured the resulting paralysis is of the lower arm type (Klumpke) There is paralysis of the small muscles of the hand, and the wrist and finger flexors *are often affected Sensory loss is found on the inner* side of the hand and forearm and there may also be a small pupil and enophthalmos on the same side Cervical ribs usually cause lesions of the lower arm *type Such a cervical rib is liable to develop from the* 7th cervical vertebra when the brachial plexus is pre fixed the C 4 root joining the plexus and the C 4 and 5 nerve roots being large whereas the *C 8 and Th 1 roots are small Often there are bilateral* cervical ribs, but symptoms are only found on one side, usually the right They are due to pressure of the rib on the lower segments of the plexus They do not usually show themselves until the patient grows up In some cases in soldiers they were first noticed when the patient wore a pack The symptoms and signs are of the lower arm type as described above There may be numbness tingling or pain on the inner side of the hand or forearm which is relieved by elevating the hand There may also be wasting of the small muscles of the hand and a claw hand may develop The radial pulse may also be weaker on the affected side In the so called *scalenus anticus syndrome* (see p 245) it is suggested that there is first drooping of the shoulder then compression of Th 1 root and the subclavian artery between the normal first rib and the clavicle This leads to scalenus anticus spasm The term *thoraco axillary inlet syndrome* is non committal as regards etiology and so preferable It is not uncommon in women who have to carry heavy shopping bags Acroparæsthesia of the hands and arms observed chiefly in middle aged and elderly women who have to perform unusual manual work may be due to the same cause The patient complains of burning pain tingling sagging of the shoulder numbness and uselessness and at times pallor or blueness of the fingers worse at night

**Differential Diagnosis** This is established by X ray examination but care should be taken to exclude other diseases causing muscle wasting of the hand such as syringomyelia and progressive muscular atrophy

**Treatment** If the cervical rib is giving rise to definite symptoms it should be removed surgically In the thoraco axillary inlet syndrome the affected arm should be rested in a sling *In more advanced cases* the subclavian artery may be freed by division of the scalenus anticus

to the palm of the hand and hypothenar eminence (b) Dorsal to the back of the wrist and hand the little finger and the inner half of the ring finger The superficial terminal branch supplies the skin of the palmar surface of the inner one and a half fingers The deep terminal branch is distributed to the following muscles The flexor brevis minimi digiti the abductor minimi digiti the opponens minimi digiti the interossei the third and fourth lumbricals the adductor obliquus and transversus pollicis and the flexor brevis pollicis

**Etiology of Lesions** The ulnar nerve may be affected by 1 Trauma such as a wound or injury of the arm or elbow 2 Neuritis due to cold or to leprosy

**Clinical Findings** If the lesion is situated near the elbow the patient notices weakness in flexing the wrist

*On Examination* There may be radial deviation and extension at the wrist joint the fingers are extended at the metacarpo phalangeal joints but flexed at the interphalangeal joints especially the two inner fingers There is inability to adduct the thumb and the patient cannot separate the fingers With a lesion at the wrist a claw hand develops there is wasting of the hypothenar eminence and of the spaces between the metacarpal bones There is also loss of sensation over the inner one and a half fingers

**Treatment** The arm should be rested and the forearm and hand supported in a splint

## The Median Nerve

**Anatomy** The median nerve is formed from the outer and inner cords of the brachial plexus from the C 5 C 6 C 7 C 8 and Th 1 nerve roots

The median nerve passes down the front of the arm on the outer side of the brachial artery and in the lower third of the arm crosses in front of the artery to its inner side It reaches the forearm between the two heads of the pronator radii teres muscle It runs down the middle of the forearm deep to the superficial muscles and enters the palm on the outer side of the flexor tendons of the fingers There are no branches in the arm In the forearm it gives off the anterior interosseous and the palmar cutaneous branches The anterior interosseous nerve gives *branches to the flexor longus pollicis and the outer half of the flexor* profundus digitorum and the pronator quadratus muscles The palmar cutaneous branch supplies the skin of the palm The median nerve in the hand supplies the following muscles The abductor and opponens pollicis the superficial head of the flexor brevis pollicis and the two outer lumbricals The cutaneous branches are distributed to both sides of the thumb the radial side of the index finger and the adjacent sides of the second and third and third and fourth fingers

**Etiology of Lesions** The median nerve may be affected by 1 Trauma such as a wound in the arm or forearm 2 Pressure as by contractions of the two heads of the pronator radii teres in playing tennis ( tennis elbow ) or continued use of the arm as in dentistry

At first the nerve lies behind the axillary artery and in th
third of the arm it passes behind the brachial artery on the inn
of the arm In the middle third of the arm it passes outwards l
the humerus, and reaches the outer side of the humerus in the
third of the arm entering the forearm by passing in front of the ext
condyle of the humerus It then divides into the radial and post
interosseous nerves The musculo spiral nerve supplies the tric
supinator longus and extensor carpi radialis longior muscles It g
an internal and external cutaneous branch the former supplying
skin over the upper third of the inner side of the arm and the latter t
skin over the lower third of the outer and back part of the arm an
the upper half of the back of the forearm and the upper and inne
two thirds of the back of the forearm

*The Radial Nerve* This supplies the skin over the back of the wrist
the outer side and back of the hand the back of the thumb and outer
two and a half fingers

*The Posterior Interosseous Nerve* This runs round the outer side of
the radius to reach the back of the forearm and travelling deep to the
extensor tendons ends on the back of the carpus It supplies the
following muscles The extensor carpi radialis brevior supinator
brevis extensor communis digitorum extensor minimi digiti extensor
carpi ulnaris extensor ossis metacarpi pollicis extensor longus pollicis
extensor brevis pollicis and the extensor indicis

**Etiology of Lesions** The musculo spiral nerve or its branches
may be affected by 1 Trauma by a fractured humerus pressure of
a crutch or by pressure from sleeping on the arm when under the influence
of alcohol 2 Neuritis due to cold or to lead

**Clinical Findings** Paralysis of the muscles supplied by the musculo
spiral nerve causes wrist drop There is inability to extend the fingers
at the metacarpo phalangeal joints or to extend the elbow or supinate
the forearm There is rarely loss or blunting of sensation over the
back of the arm forearm and outer side of the back of the hand

**Treatment** The forearm and hand should be supported on a
plint with the wrist and fingers slightly extended

## The Ulnar Nerve

**Anatomy** The ulnar nerve is formed from the C 8 and Th 1
ve roots from the inner cord of the brachial plexus It passes in
axilla between the axillary artery and vein In the upper half
e arm it lies in front of the triceps muscle internal to the brachial
y At the elbow it is placed between the internal condyle of
umerus and the olecranon process of the ulna In the forearm
between the flexor carpi ulnaris and flexor profundus digitorum
s and passing in front of the anterior annular ligament into
lm of the hand it divides into a superficial and deep branch
re no branches in the upper arm In the forearm the following
s arise Muscular to the flexor carpi ulnaris and inner half
lexor profundus digitorum muscles Cutaneous (a) Palmar

narrowing which limit flexion and extension of the spine and shorten the spine The intervertebral foramina are thus narrowed from above downwards and the nerve roots may be kinked The disc may protrude or it may rupture Pressure may thus occur on the nerve in the foramen on the spinal nerve root or on the cord Osteophytes may form on the neuro central joints partially obliterating the inter vertebral foramina or intraspinally compressing the cord or on the anterior surface of the body of the vertebra impinging on the œsophagus Finally fibrotic changes may compress the nerve roots and radicular nerves In some cases the vertebræ become fused in others they are unduly mobile with spondylolisthesis

Clinical Findings A disc may rupture suddenly causing severe pain in the neck with limitation of movement Usually the onset is insidious with *nerve root symptoms* Pain radiates to the shoulder arm hand digits and may be felt in the upper interscapular region or in the upper part of the front of the chest The trapezius muscle may go into spasm on one side In some cases pain radiates to the side of the neck to the teeth or to the ears Paræsthesia is often an early symptom burning or tingling sensations in the fingers or there may be numbness and difficulty in picking up small objects The radial side of the arm and hand is more often affected than the ulnar Pressure of osteophytes on the œsophagus may cause dysphagia Some patients complain of hoarseness when tired Muscular wasting of the arms or hands is not common The symptoms are markedly aggravated by flexing the neck and by vibration when in a car or train

*Spinal cord symptoms* (myelopathy) Compression of the cord affects the legs more often than the arms but all four limbs may be involved in a quadriplegia Paræsthesia is an early symptom in the feet with a cotton wool sensation This leads to unsteadiness and the patient may stand on a wide base and like to have one leg touching a solid object to gain support Spastic weakness may develop with root pains in the legs The deep reflexes in the arms are often increased and usually some slight sensory loss can be detected

In all cases of suspected cervical spondylosis the cervical spine must be X rayed obliquely to show the intervertebral foramina laterally to show narrowing of the discs and antero posteriorly In some cases a myelogram is required to show a disc protrusion or obliteration of the nerve root pouch in the intervertebral foramen

Differential Diagnosis Cervical spondylosis produces such protean symptoms and signs that many diseases may be simulated and must be considered in establishing the diagnosis These include brachial neuritis angina pectoris progressive muscular atrophy the scalenus anticus syndrome disseminated sclerosis subacute combined degeneration of the cord a spinal cord tumour or even a mid brain tumour X ray *examination of the cervical spine usually reveals* the nature of the trouble although in many people there are X ray appearances of cervical spondylosis without the patient noticing any symptoms

Prognosis This varies with the nature of the lesion Cases diagnosed

3 The median nerve may be compressed in the carpal tunnel Th
liable to occur in women from pressure on the wrists as in scrubbin
following fracture It is often bilateral

*Clinical Findings* *If the lesion occurs above the elbow ther*
weakness in pronating the forearm and in flexing the wrist and
interphalangeal joints The thumb also cannot be abducted '
hand is deviated to the ulnar side When the lesion is located at
wrist there is wasting of the thenar eminence and inability to abduct
thumb Loss of sensation in both lesions is found over the thumb
the two and a half outer fingers The patient often complains of sev
burning pain (causalgia) in the hand when the nerve is incomplet
divided Trophic changes appear in the hand, redness sweating
glossy skin, overgrowth and curving of the nails wasting of the fin
*pads swelling of the joints and fragility of bones When the nerve*
compressed in the carpal tunnel there is weakness and wasting of
abductor brevis and opponens pollicis There may also be pain a
tingling in the region of the cutaneous supply of the thumb and finge

**Treatment** The arm should be rested in a sling and the thu
muscles prevented from stretching by an adequate splint Division
the transverse carpal ligament will relieve carpal tunnel compressi
Procaine block of the sympathetic or sympathectomy may be requir
for causalgia

## The Musculo cutaneous Nerve

**Anatomy** The musculo cutaneous nerve arises from the out
cord of the brachial plexus from the C 5 and C 6 nerves

The musculo cutaneous nerve passes down the arm between t
biceps and brachialis anticus muscles to the elbow It supplies branch
to these two muscles and to the coracobrachialis In the forearm
supplies the skin on the outer and posterior aspect

*Etiology of Lesions The nerve may be injured by trauma*
involved in a neuritis

**Clinical Findings** The patient has difficulty in flexing the forearm
and sensation may be lost over the outer side and back of the forearm

## Cervical Spondylosis

**Definition** A condition characterised by degenerative changes in
the cervical intervertebral discs and vertebræ accompanied by cervical
radiculitis and at times by cervical cord compression

**Etiology** The cause is unknown In some cases cervical spondylosis
follows trauma to the neck which may not have been *recognised at the*
time It may be due to whiplash injury to the neck caused usually by
a car collision from behind or it may follow straining of the neck under
an anæsthetic

**Pathology** The most important lesion is compression of the cervical
nerves in the intervertebral foramina Less often the nerve roots are
compressed before they enter the foramina or the spinal cord is com
pressed The pathological changes *include disc degeneration* and

narrowing which limit flexion and extension of the spine and shorten the spine The intervertebral foramina are thus narrowed from above downwards and the nerve roots may be kinked The disc may protrude or it may rupture Pressure may thus occur on the nerve in the foramen on the spinal nerve root or on the cord Osteophytes may form on the neuro central joints partially obliterating the inter vertebral foramina or intraspinally compressing the cord or on the anterior surface of the body of the vertebra impinging on the œsophagus Finally fibrotic changes may compress the nerve roots and radicular nerves In some cases the vertebræ become fused in others they are unduly mobile with spondylolisthesis

**Clinical Findings** A disc may rupture suddenly causing severe pain in the neck with limitation of movement Usually the onset is insidious with *nerve root symptoms* Pain radiates to the shoulder arm hand digits and may be felt in the upper interscapular region or in the upper part of the front of the chest The trapezius muscle may go into spasm on one side In some cases pain radiates to the side of the neck to the teeth or to the ears Paræsthesia is often an early symptom burning or tingling sensations in the fingers or there may be numbness and difficulty in picking up small objects The radial side of the arm and hand is more often affected than the ulnar Pressure of osteophytes on the œsophagus may cause dysphagia Some patients complain of hoarseness when tired Muscular wasting of the arms or hands is not common The symptoms are markedly aggravated by flexing the neck and by vibration when in a car or train

*Spinal cord symptoms* (myelopathy) Compression of the cord affects the legs more often than the arms but all four limbs may be involved in a quadriplegia Paræsthesia is an early symptom in the feet with a cotton wool sensation This leads to unsteadiness and the patient may stand on a wide base and like to have one leg touching a solid object to gain support Spastic weakness may develop with root pains in the legs The deep reflexes in the arms are often increased and usually some slight sensory loss can be detected

In all cases of suspected cervical spondylosis the cervical spine must be X rayed obliquely to show the intervertebral foramina laterally to show narrowing of the discs and antero posteriorly In some cases a myelogram is required to show a disc protrusion or obliteration of the nerve root pouch in the intervertebral foramen

**Differential Diagnosis** Cervical spondylosis produces such protean symptoms and signs that many diseases may be simulated and must be considered in establishing the diagnosis These include brachial neuritis angina pectoris progressive muscular atrophy the scalenus anticus syndrome disseminated sclerosis subacute combined degeneration of the cord a spinal cord tumour or even a mid brain tumour X ray examination of the cervical spine usually reveals the nature of the trouble although in many people there are X ray appearances of cervical spondylosis without the patient noticing any symptoms

**Prognosis** This varies with the nature of the lesion Cases diagnosed

early usually respond quickly to treatment, more severe ones m
to invalidism for months or years

**Treatment** Mild cases may be treated by wearing a plastic c
giving up work More severe cases should be put to bed and the
and the patient may make a complete symptomatic recovery wi
immobilised between sandbags day and night There should onl
one pillow and a fracture board should be used A plastic collar sho
be worn intermittently for 3 to 6 months when the patient gets
especially on travelling in a car or train, or on doing work involv
flexion of the neck Neck traction is undesirable and may lead
severe giddiness lasting for several months after traction is discontinued
This may be due to disturbance of circulation in the vertebral arteries
which run through the foramina in the transverse processes of the
upper six cervical vertebræ The posterior inferior cerebellar artery
and the anterior spinal artery are branches of the vertebral artery and
so the cerebellar and spinal cord circulation may be affected Operation
is required in some cases for removal of osteophytes and decompression
of the spinal or radicular nerves

## The Lumbo sacral Plexus and its Branches

**Anatomy** The lumbo sacral plexus is formed from the anterior primary divisions of the 1st to 5th lumbar and 1st to 5th sacral nerves

## The Anterior Crural Nerve

**Anatomy** The anterior crural nerve is derived from the L 2 L 3 and L 4 nerves It supplies muscular branches to the iliacus pectineus sartorius and quadriceps extensor muscles The sensory branches are the middle and internal cutaneous nerves passing to the anterior and inner side of the thigh and the internal saphenous nerve supplying the inner side of the leg foot and big toe

**Etiology of Lesions** The anterior crural nerve may be affected by
Trauma in fracture of the pelvis or femur or by wounds in the groin
Pressure due to a prolapsed intervertebral disc between L 3 and
4 to a psoas abscess a tumour of the pelvis enlarged inguinal
ands or an aneurysm of the iliac artery 3 Arthritis of the spine or
4 Neuritis due to diabetes mellitus 5 Neuralgia associated
th chronic constipation

**Clinical Findings** An organic lesion of the anterior crural nerve
es rise to weakness of the thigh, the patient complaining that his
e lets him down

*On Examination* There is weakness and wasting of the extensors
e knee and slightly of the hip flexors The affected muscles are
d Tenderness may be found on pressure on the inner side of the
The knee jerk is diminished or absent, and the quadriceps
or muscle may show a reaction of degeneration There may be
esia or paræsthesia over the inner side of the thigh or leg In
neuralgia there is pain on the inner side of the thigh or leg and

the inner side of the foot In all cases a rectal examination should be made to exclude a growth together with an X ray examination of the pelvis and spine

**Prognosis and Treatment** These vary with the cause In cases due to neuritis the treatment consists in rest and light massage Support may be given to the knee by an elastic band passing down the front of the thigh and attached above to a belt on the thigh and below to a garter around the calf

## The Obturator Nerve

**Anatomy** The obturator nerve is derived from the L 2 L 3 and L 4 nerves The superficial branch supplies the adductors of the thigh and the skin over the inner part of the lower two thirds of the thigh The deep branch supplies the obturator externus muscle (an external rotator of the thigh) and the knee joint

**Etiology of Lesions** The nerve may be injured in women during *labour or by the pressure of a pelvic tumour or an obturator hernia*

**Clinical Findings** In obturator neuralgia there is pain over the inner side of the thigh When the nerve is definitely injured by pressure there is weakness of the thigh adductors as shown by difficulty in crossing the leg and weakness in external rotation of the hip may also be detected There may be blunting of sensation over the lower part of the inner side of the thigh

**Treatment** This varies with the cause

## The External Cutaneous Nerve

**Anatomy** The external cutaneous nerve is derived from the L 2 and L 3 nerves It enters the thigh close to the anterior superior iliac spine beneath the outer end of Poupart s ligament It is a sensory nerve which supplies the skin on the outer side of the thigh and below the great trochanter of the femur

**Etiology of Lesions** Lesions may be due to trauma to pressure in pregnancy to neuritis or to local fibrositis in the tunnel of the fascia lata

**Clinical Findings** Neuritis of the external cutaneous nerve is known as *meralgia paræsthetica* It is generally caused by tension of the overlying fascia It usually affects obese middle aged men It begins with a sense of numbness over the antero lateral aspect of one thigh Later there is burning tingling and pain worse on standing or walking A tender spot may be found at the outer end of Poupart s ligament where the nerve enters the thigh

**Treatment** In some cases it is advisable to excise the nerve to relieve pain

## The Sciatic Nerve

**Anatomy** The sciatic nerve is derived from the L 4 L 5 S 1 S 2 and S 3 nerves The nerve before division supplies the ham string muscles which flex the knee (the biceps femoris semimem branosus semitendinosus and a portion of the adductor magnus)

early usually respond quickly to treatment, more severe ones may l to invalidism for months or years

Treatment Mild cases may be treated by wearing a plastic coll and the patient may make a complete symptomatic recovery with giving up work More severe cases should be put to bed and the he immobilised between sandbags day and night There should only one pillow and a fracture board should be used A plastic collar shou be worn intermittently for 3 to 6 months when the patient gets u especially on travelling in a car or train, or on doing work involvi flexion of the neck Neck traction is undesirable and may lead severe giddiness lasting for several months after traction is discontinue This may be due to disturbance of circulation in the vertebral arteri which run through the foramina in the transverse processes of t upper six cervical vertebræ The posterior inferior cerebellar arte and the anterior spinal artery are branches of the vertebral artery a so the cerebellar and spinal cord circulation may be affected Operati is required in some cases for removal of osteophytes and decompressi of the spinal or radicular nerves

## The Lumbo sacral Plexus and its Branches

**Anatomy** The lumbo sacral plexus is formed from the anteri primary divisions of the 1st to 5th lumbar and 1st to 5th sacral nerve

## The Anterior Crural Nerve

**Anatomy** The anterior crural nerve is derived from the L 2 L and L 4 nerves It supplies muscular branches to the iliacus pectineu sartorius and quadriceps extensor muscles The sensory branches ar the middle and internal cutaneous nerves passing to the anterior an inner side of the thigh and the internal saphenous nerve supplying th inner side of the leg foot and big toe

**Etiology of Lesions** The anterior crural nerve may be affected by 1 Trauma in fracture of the pelvis or femur or by wounds in the groin 2 Pressure due to a prolapsed intervertebral disc between L 3 and L 4 to a psoas abscess a tumour of the pelvis enlarged inguina glands or an aneurysm of the iliac artery 3 Arthritis of the spine o hip 4 Neuritis due to diabetes mellitus 5 Neuralgia associated with chronic constipation

**Clinical Findings** An organic lesion of the anterior crural nerve gives rise to weakness of the thigh the patient complaining that his knee lets him down

*On Examination* There is weakness and wasting of the extensors of the knee and slightly of the hip flexors The affected muscles are flaccid Tenderness may be found on pressure on the inner side of the thigh *The knee jerk is diminished or absent* and the quadriceps extensor muscle may show a reaction of degeneration There may be anæsthesia or paræsthesia over the inner side of the thigh or leg In crural neuralgia there is pain on the inner side of the thigh or leg and

## Sciatica

*Definition* Three conditions are commonly included by the term sciatica 1 Interstitial neuritis of the sciatic nerve 2 Symptomatic sciatica in which the nerve is involved by compression or by extension of an inflammatory process 3 Pain referred along the sciatic nerve which itself is not involved in any pathological process

Etiology Some authorities deny the existence of sciatic neuritis but interstitial neuritis may be due to some toxi infective condition such as focal sepsis rheumatism alcoholism diabetes mellitus syphilis etc Symptomatic sciatica is commonly due to fibrositis spreading from adjacent muscles and fasciæ to involve the sheath of the sciatic nerve Other causes include spinal cord tumour trauma spinal caries spondylitis meningitis fractured pelvis prolapsed inter vertebral disc a pelvic tumour etc It does not follow that osteo arthritis of the spine as revealed by X ray examination is necessarily the cause of sciatica in an individual case Pain may be referred reflexly along a healthy sciatic nerve from osteoarthritis of the hip sacro iliac strain etc *Predisposing causes* 1 Age Usually over 20 years 2 Sex Males predominate 3 Strain of the muscles of the back 4 Chill as by sitting on wet grass

Clinical Findings In the type which is secondary to fibrositis of the lumbar and gluteal muscles the onset is usually insidious The patient is an adult who gives a history of straining his back as by digging or by lifting Pain and stiffness are felt across the small of the back The patient may pay no attention to it and strain his back still further Indications that the inflammation has spread to the sheath of the sciatic nerve are afforded later perhaps after 1 or 2 months There is a sensation of numbness or tingling often felt in the heel when the foot is first put to the ground after resting This may quickly pass off and recur from time to time Further on sitting a sensation of numbness may be felt in the back of the thigh or in the leg as if the leg has gone to sleep Later pain may be experienced either in the back of the thigh or in the leg This may also be transient and not give rise to any anxiety If the disease progresses the pain becomes more acute and more persistent The patient finds that he cannot sit on any ordinary chair with comfort the weight is put on the sound side and a soft cushion is welcomed On lying he is usually free from pain but twitching or cramps may occur in the calf muscles and disturb sleep Later the pain may be more acute and disturb sleep The patient then usually prefers to lie on his back with the hip and knee flexed and the ankle plantar flexed It is very difficult for him to turn over on his face owing to pain If he lies on his side it is usually on the affected side because lying on the sound side causes a drag in the gluteal region on the affected side Walking now becomes difficult the hip and knee are kept slightly flexed and the heel is not put to the ground The pain in sciatica can be very acute of a deep seated burning and boring nature causing considerable exhaustion

*On Examination* In a developed case definite signs are present *Spinal motor system* The affected leg (*a*) Power This is usually

It divides into the external and internal popliteal nerves *The external popliteal nerve (peroneal nerve)* This passes down from the lower part of the thigh to the back of the head of the fibula where it divides into the anterior tibial and musculo cutaneous nerves *The anterior tibial nerve* supplies the ankle flexor muscles and the extensor muscles of the toes (i e, the tibialis anticus extensor longus digitorum and peroneus tertius muscles) It also gives a cutaneous branch to the skin between the great and second toes *The musculo cutaneous nerve* supplies the peroneus longus and brevis muscles (which evert and extend the ankle), and cutaneous branches to the skin over the front of the lower part of the leg, the dorsum of the foot the inner side of the great toe and the adjacent sides of the 2nd, 3rd 4th and 5th toes *The internal popliteal nerve (tibial nerve)* This is known as the posterior tibial nerve when it enters the back of the leg (at the lower border of the popliteus muscle) It divides at the ankle into the internal and external plantar nerves It supplies branches to the calf muscles which extend the ankle and flex the toes (i e, the gastrocnemius soleus, plantaris popliteus, tibialis posticus, flexor longus digitorum and flexor longus hallucis) and cutaneous branches to the outer side of the back of the leg the heel and back of the sole of the foot *The internal plantar nerve* This supplies muscular branches to the abductor hallucis, flexor brevis hallucis flexor brevis digitorum and first lumbrical muscles and cutaneous branches to the inner side of the sole of the foot and the plantar surface of the inner three and a half toes *The external plantar nerve* This supplies the remaining small muscles of the foot and cutaneous branches to the outer side of the sole of the foot, and the plantar surface of the outer one and a half toes

**Etiology of Lesions** The sciatic nerve may be affected by inflammatory changes of the spine or meninges at its origin from the cord, by a spinal tumour or prolapsed intervertebral disc In the pelvis it may be involved in a fractured pelvis or by a tumour or the pregnant uterus At the sciatic notch or in the thigh it may be injured by a wound or involved by inflammation of its sheath The external popliteal nerve is liable to injury and it may be affected in lead neuritis The internal popliteal nerve is rarely affected

**Clinical Findings** *Lesions in the pelvis or thigh* These result in paralysis of the hamstrings and all the muscles below the knee There is inability to flex the knee and the foot is dropped The patient can neither stand on his heel nor on his toes, but he can usually walk There is loss of the ankle jerk and anæsthesia is found over the foot and the lower two thirds of the calf *Lesions of the external popliteal nerve* The foot is dropped and inverted the toes are flexed and cannot be extended The patient can stand on his toes There is anæsthesia on the outer half of the front of the leg and on the dorsum of the foot and adjacent sides of the toes *Lesions of the internal popliteal nerve* The patient cannot extend his foot and cannot flex the toes He cannot stand on his toes There is anæsthesia on the lower third of the outer and back part of the leg, and the sole of the foot and plantar surface of the toes Later contracture may occur causing a claw foot

insulin and a correct diet. Local treatment consists in the application of heat to the leg either by hot bottles, or by diathermy. If tender "fibrositic" nodules are found in the gluteal muscles they should be injected with 20 to 40 ml. of 1% procaine. The injection may be repeated next day and again after a day's interval, and in some cases the sciatic pain is quickly relieved. No violent massage or nerve stretching should be employed, as this only does harm and aggravates the sciatica. If the pain is severe the affected limb should be immobilised in plaster. Analgesics such as aspirin gr. 10 to 15 or tab. codein. co. nocte will usually secure sleep. High sciatica may be treated by epidural injections if other measures fail. These consist in the injection through the sacro-coccygeal foramen of 20 ml. of 1% procaine solution and 60 ml. of normal saline. The patient should lie down for a day or so after the injection.

## Prolapsed Intervertebral Disc

### (*Herniated Nucleus Pulposus*)

**Definition.** Backward displacement of portions of an intervertebral disc.

**Etiology.** Since 1934 interest has been increasingly directed to a condition of backward displacement of a ruptured intervertebral disc. A history of trauma to the back is given in about 80% of cases. It is claimed to be the cause of sciatic pain in many cases diagnosed as sciatica.

**Pathology.** There is rupture of the annulus fibrosus of a disc and extrusion of the nucleus pulposus. On rupture portions protrude into the spinal canal and may cause pressure on a nerve root. The discs most frequently affected lie between L. 4 and L. 5 or L. 5 and S. 1 vertebræ. Less frequently the lesion occurs in the cervical or thoracic region.

**Clinical Findings.** The patient is usually an adult about the age of 40 who complains of recurring attacks of pain in the lower part of the back, followed by intractable pain in the buttock, posterior part of the thigh and postero-lateral region of the calf. Tingling or pricking sensations may be felt on the outer side of the foot. The onset may be sudden, following lifting a heavy weight with the spine flexed.

*On Examination.* Bending forwards is restricted, bending sideways is free unless there is scoliosis. In sciatic scoliosis which is usually due to a disc lesion, bending is free to the side which increases the deformity. Tenderness may be elicited over L. 4 or L. 5 spines and over the sciatic nerve in the thigh. There is pain on stretching the sciatic nerve. The ankle jerk is absent in about 50% of cases and there may be diminution of cutaneous sensation on the outer side of the foot. In some cases the bladder is paralysed. If the lesion is in the cervical or thoracic region death may result from paralysis due to destruction of the cord. If the disc protrudes between L. 3 and L. 4 the pain will be in the region of the anterior crural nerve and extend down to the medial malleolus. The cerebro-spinal fluid contains a slight excess of protein in about two-thirds of the cases with a normal cell count. These symptoms and signs

normal but some weakness of the knee flexors or leg muscles may develop later (*b*) Tone There is flaccidity of the affected muscles (*c*) Wasting This is often present in the calf and thigh and the gluteal fold is much diminished (*d*) Contractions Fibrillation and cramp may occur in the calf muscles

*Spinal sensory system* Complete anæsthesia is rarely found but there may be blunting of sensation on the dorsum or side of the foot or on the outer side of the leg Tender points may be found along the course of the sciatic nerve in the back of the thigh, or in the calf There is pain on stretching the sciatic nerve, when the hip is flexed with the knee extended (Lasègue s sign) There is no pain on abducting or externally rotating the hip with the knee flexed

*The Reflexes* The knee jerk is normal or exaggerated The ankle jerk is often diminished or lost The plantar reflex is normal or absent

Examination of the back with the patient lying prone on a cushion placed under the lower part of the abdomen often reveals the presence of the so called fibrous nodules in the lumbar and gluteal muscles These are thought to be lipomatous protrusions through the fascia and are tender on pressure In cases due to a pelvic tumour, rectal examination may reveal the cause and this should be carried out in every case of sciatica A sarcoma of the ilium may be obvious on external examination of the back An X ray examination should always be made of the spine and pelvis including the hip joint, as in this way arthritis may be detected The urine should also always be tested for glucose Bilateral sciatica is very suggestive of a pelvic tumour, but it may occur in diabetic or in fibrositic sciatica In some cases fibrositic sciatica has an acute onset

**Differential Diagnosis** Some authorities consider that a severe case of sciatica as described above is due to a prolapsed disc but in the latter the pain is usually more acute at the onset and the back may feel as if it is broken across In arthritis of the hip there is some limitation of movement of the joint and the X ray findings are usually diagnostic Prolapse of an intervertebral disc is considered on p 459

**Course and Complications** Sciatica usually pursues a prolonged course lasting several months or 1 or 2 years but the ultimate tendency is to recovery Recurrences undoubtedly occur in the same leg After a severe attack the sciatic nerve remains a weak spot in the body and any violent strain may provoke another attack Permanent muscular wasting and diminution of the ankle jerk are liable to remain The course in pressure sciatica is progressive unless the cause is removed

**Prognosis** The outlook is usually good in sciatica but in pressure sciatica the prognosis is generally unfavourable unless it is due to a simple tumour which can be removed

**Treatment** The sooner the patient takes to his bed the sooner a fibrositic sciatica will be cured Constipation must be adequately treated The teeth should be X rayed and if any apical infection is present it should be treated The blood Wassermann reaction should be determined and if positive a course of anti syphilitic treatment should be given If diabetes mellitus is found it will be necessary to administer

insulin and a correct diet Local treatment consists in the application of heat to the leg either by hot bottles or by diathermy If tender ' fibrositic nodules are found in the gluteal muscles they should be injected with 20 to 40 ml of 1% procaine The injection may be repeated next day and again after a day s interval and in some cases the sciatic pain is quickly relieved No violent massage or nerve stretching should be employed as this only does harm and aggravates the sciatica If the pain is severe the affected limb should be immobilised in plaster Analgesics such as aspirin gr 10 to 15 or tab codein co nocte will usually secure sleep High sciatica may be treated by epidural injections if other measures fail These consist in the injection through the sacro coccygeal foramen of 20 ml of 1% procaine solution and 60 ml of normal saline The patient should lie down for a day or so after the injection

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are very suggestive of a prolapsed disc X ray examination of the spine may show narrowing of a disc space Myelography is usually inadvisable and unnecessary in order to establish the diagnosis

Treatment Doubtless many cases recover with the usual treatment of sciatica, rest in bed and warmth If, however, the condition proves intractable the protruding portions of the disc may be removed by laminectomy In some cases the relief of pain is dramatic in others the post-operative convalescence is slow and the results may be very un satisfactory Post operative complications are not unknown preventing the patient from returning to work, and deaths have occurred from wound or bladder sepsis directly resulting from the operation

## CHAPTER V

# THE URINARY SYSTEM

**Introductory** Clinical examination in diseases of the urinary system implies far more than an examination of the urine The cardio vascular system the blood and the tissue fluids are intimately linked with this excretory system Information will be required on the following points *The urine* The colour reaction and odour The amount and specific gravity, by day and night The consistency and deposit *Chemical examination* Proteins albumin globulin Bence Jones protein albumose and hæmoglobin derivatives such as methæmoglobin and porphyrin Bile salts and pigment Reducing substances such as glucose lactose pentose creatinin uric acid and homogentisic acid Chlorides *Microscopical examination* Blood cells (red and white) pus cells renal or vesical epithelium casts crystals phosphates uric acid urates oxalates cystine leucine and tyrosine In acid urine the following crystalline substances may be found Uric acid sodium urate hippuric acid calcium oxalate cystine xanthine leucine and tyrosine In alkaline urine crystals include calcium phosphate (stellar phosphate) ammonium magnesium phosphate ammonium urate calcium carbonate and cholesterin Bacteria such as the Bact coli the Mycobacterium tuberculosis (B tuberculosis) staphylococci and streptococci Ova such as those of the bilharzia *The blood* Chemical examination Total protein albumin and globulin urea non protein nitrogen cholesterol and chlorides Blood counts Wassermann reaction *The cardio vascular system* The size of the heart valvular or myocardial lesions the condition of the aorta and peripheral vessels the blood pressure and ophthalmoscopic examination

### Abnormalities of the Urine

**Amount** The amount of urine passed in the 24 hours by an adult averages 50 oz —37 by day and 13 by night This volume may be increased or decreased

**Polyuria** This is a symptom of many conditions such as Nervousness cold over drinking diuretics such as tea beer etc diseases such as diabetes insipidus diabetes mellitus chronic nephritis and hyperpiesis

**Oliguria and Anuria** Oliguria may be due to various causes such as Scanty intake of fluids exercise sweating hysteria acute nephritis and fevers Anuria may result from 1 Suppression no urine being formed This may be due to the acute or the terminal stages of nephritis severe shock and collapse as in coronary thrombosis or cholera and operations such as passing a catheter It may also result from shock due to operations or hæmorrhage or be due to a mis matched blood transfusion a soap induced abortion or mercury poisoning 2 Obstruc

tion This may be due to stricture an enlarged prostate carcinoma of the bladder blocking the orifices of the ureters calculi in both ureters, a calculus obstructing one ureter the other kidney having been removed or removal of the sole functioning kidney

**Specific Gravity** Normally this varies by day and night being by day 1 018 and by night 1 026 The specific gravity of a single specimen is valueless as a clinical guide at least a 12 hours specimen is required

**Colour and Consistency** The colour may be pale in a low specific gravity urine as in chronic nephritis or in a high specific gravity urine as in diabetes mellitus The urine may be turbid or show a deposit due to Urates, phosphates, mucin, pus and organisms Various colours may be noted, such as Brown, due to bile or to urates red owing to blood or its derivatives or to Pyridium when the urine is acid

Some individuals have an idiosyncrasy towards beetroot and pass red urine 4 to 6 hours after taking it The urine is clear turns yellow with alkali and red again with hydrochloric acid No absorption band is seen spectroscopically The fæces may be red or purple and there may be diarrhœa

The urine *may be bright yellow, due to riboflavin black* due to melanin carbolic acid and alkapton white from chyle green from bile methylene blue and carbolic acid pink from a deposit of urates blue due to methylene blue orange from excess of urobilin santonin *or rhubarb The urine may be frothy due to gas*

## Albuminuria

## (*Proteinuria*)

The clinical significance of albuminuria cannot be determined accurately by any test for renal function There are two groups of cases, benign and organic

*Benign Albuminuria (functional physiological postural cyclical* orthostatic) This is liable to occur in growing children, especially boys At one school it was present in 10% of the boys It is often associated with lordosis The morning specimen is free from albumin and globulin but *samples collected after the patient has been up* and about contain a small quantity of protein up *to 1% In some cases* there is more globulin than albumin Further the specimen passed at the end of the day usually contains less than that passed after the patient *has been up for only an hour or so It should be noted* that the morning specimen may contain a trace of albumin owing to the continued elimination of albumin for half an hour or so after the patient first goes to bed This apparent anomaly can be recognised if the *bladder is emptied halfway through the night*, when the specimen passed on rising will be free from albumin The urine contains no casts or only an occasional hyaline or granular one and oxalate crystals may be present There are no cardio vascular changes the blood pressure *is normal* there *is no* œdema and the renal function tests are normal The cause of the condition is not known but the patient usually grows out of it although it may last in about one fifth of

the cases until the age of 30 It has been suggested that it is due to lordosis It does not lead to subsequent renal disease Administration of calcium lactate gr 10 to 15 or of sodium bicarbonate gr 60 t d s may cause the albuminuria to disappear temporarily Violent exercise such as rowing a course leads to a temporary albuminuria in the majority of adult athletes Postural albuminuria may also occur after infections such as scarlet fever, no albumin being passed as long as the patient remains in bed

**Organic Albuminuria** *Renal causes* Nephritis nephrosis passive congestion (cardiac kidney) infarction, tuberculosis, polycystic disease malignant tumours amyloid degeneration papilloma of the renal pelvis pyelitis and calculus *Ureteric causes* A stone *Vesical causes* Cystitis new growths calculus and tuberculosis *Urethral causes* Urethritis and prostatitis

Febrile albuminuria may occur in any condition in which there is moderately high pyrexia, and is probably a manifestation of the toxæmic kidney (see p 480)

## Albumosuria

This may appear during the resolution period of lobar pneumonia in acute yellow atrophy of the liver during the puerperium or in chronic suppuration Bence Jones protein is associated with multiple myelomata

## Phosphaturia

The urine is alkaline The phosphates may only be seen on boiling, or they constitute a whitish opacity often noticed at the end of micturition and mistaken by the male patient for semen In such instances depression neurasthenia or wasting may be noted The condition is often associated with cystitis due to a staphylococcus infection or it may result from overdosage with alkalis in the treatment of peptic ulcer or from a vegetable diet A specimen removed under aseptic conditions should be examined microscopically and the presence of crystals and organisms determined

**Treatment** This consists in giving acid sodium phosphate in doses sufficient to keep the urine acid as much as gr 60 t d s may be required Sulphonamides or antibiotics according to the sensitivity of the organism may also be given A diet poor in calcium containing substances is recommended milk and eggs being avoided

## Oxaluria

This is detected on microscopical examination of the urine It is usually of no clinical significance but in some instances it is associated with recurrent hæmaturia or with pain suggesting renal calculus or spinal disease Colonic flatulence due to carbohydrate indigestion may be the chief symptom

**Treatment** Diet Strawberries rhubarb tomatoes beetroot spinach cabbage radishes cauliflower eggs and milk should be avoided as they are rich in oxalates and lime The following substances which contain magnesium salts may be taken Potatoes bread meat apples

and peas The diet should be rich in protein and excess of carbohydrate should be avoided especially root vegetables and porridge Acid sod phosph gr 80 to 60 t d s and mag sulph gr 60 to 120 should be given in the morning Hard water should not be drunk

## Glycosuria

The following substances which reduce Fehling s solution may be found in the urine Glucose, lactose pentose homogentisic acid urates and creatinin Benedict s solution is only reduced by glucose *lævulose lactose* pentose and homogentisic acid Glucose is of the greatest clinical importance and is considered under diabetes mellitus

## Acetonuria

This is met with in acidosis, associated with diabetes mellitus, Von Gierke s disease prolonged vomiting and starvation

## Indicanuria

This can only be detected by chemical tests, an excess is indicative of intestinal stasis

## Hæmaturia

**Definition** The presence of red blood cells in the urine

***Etiology*** *The causes are very numerous and may be thus sub* divided 1 *Renal causes* Nephritis infarction tuberculosis, calculus, crystals such as oxalates or the acetyl derivatives of sulphonamides, turpentine cantharides, carbolic acid congestion movable kidney pyelitis injury, papilloma neoplasms polyarteritis nodosa and polycystic disease 2 *Ureteric causes* Calculus 3 *Vesical causes* Calculus papilloma carcinoma tuberculosis, bilharziasis acute cystitis arteriosclerosis, and varicose veins 4 *Urethral causes* Trauma, urethritis ulcerating growths and caruncle 5 *Prostatic causes* Enlargement and growths 6 *Pre renal causes* Leukæmia, purpura scurvy, *hæmophilia a low plasma prothrombin content malignant* varieties of small pox and scarlet fever

**Clinical Findings** The colour of the urine varies with the amount of blood present, it may be almost black *or present a smoky appear* ance or show a dark ring on top In other cases it is faintly tinged With small amounts of blood there are no naked eye changes but the presence of blood cells is detected microscopically If the blood is intimately mixed with the urine on passing it is probably derived from the kidneys if it appears only at the beginning of micturition it probably comes from the urethra and if it is noticed at the end of *micturition the source may be in the bladder A case of* hæmaturia may be investigated on the following lines (1 General examination of the patient for fevers purpura and scurvy (2) Examination of the heart and arteries in cases of nephritis (3 Examination of the urine Amount casts, crystals cells organisms and ova This will yield suggestive information as to nephritis oxaluria pyuria infections such as tuber culosis pyelitis, cystitis, and bilharziasis (4 Examination of the blood

Blood count blood culture Wassermann reaction plasma prothrombin concentration blood urea and non protein nitrogen These tests will be of help in leukæmia, septicæmia syphilis and chronic nephrosclerosis (5) X ray examination for stone in the kidneys ureter or bladder (6) Cystoscopy and catheterisation of the ureters and (7) pyelography This will show whether the blood is coming from the bladder or from one or both ureters Blood coming from one kidney may mean a very early stage of chronic nephrosclerosis or an angioma at the apex of a pyramid in addition to other causes such as tuberculosis tumours calculi etc Pyelography is of value in indicating the presence of a tumour deforming the pelvis of the kidney or ureter or a hydronephrosis Despite all these investigations in some cases the cause of bleeding cannot be determined and such cases are known as renal epistaxis or the essential hæmaturia of Gull Certain of these cases are due to local purpuric lesions in the kidney or renal tract

**Treatment** This is considered under the various causative conditions

## Hæmoglobinuria

**Definition** The presence of blood pigments especially methæmoglobin in the urine

**Etiology** Hæmoglobinuria results from intravascular hæmolysis This may be due to 1 Chemical substances such as potassium chlorate arsine muscarine sulphonamide quinine and Myanesin 2 Infective agents such as cause syphilis malaria (blackwater fever) yellow fever and gas gangrene due to the Clostridium welchii 3 Mismatched blood transfusions 4 Fabism idiosyncrasy to the broad bean (*vicia faba*) 5 Pregnancy and the puerperium 6 Extensive burns 7 Lederer s anæmia The cause of this is unknown 8 Spider bites A case has been recorded in America resulting from the bite of a large brown spider 9 Paroxysmal hæmoglobinuria results from (*a*) exposure to cold It may occur in viral pneumonia due to cold agglutinins in the blood (*b*) muscular exercise or standing in a position of lordosis (*c*) hæmolytic anæmia producing nocturnal hæmoglobinuria (the Marchiafava Micheli syndrome) (*d*) paralytic hæmoglobinuria or paroxysmal myoglobinuria

The primary abnormality in the Marchiafava Micheli syndrome lies in the red corpuscles as shown by the acid serum test of Ham A certain number of the patient s red cells are hæmolysed when placed in fresh untreated serum obtained either from the patient or from a normal individual Acid or carbon dioxide is added to the serum to counteract the alkalinity resulting from loss of CO when the serum is exposed to air Normal corpuscles are not hæmolysed by the patient s serum

Myoglobinuria is a rare disease in man characterised by muscular atrophy and recurrent attacks of myoglobinuria It is also met with in the crush syndrome It occurs commonly in horses which are taken out to work after a rest Myohæmoglobin derived from the muscles appears in the urine

Spectroscopic examination of the urine reveals the presence and

nature of the pigment In paroxysmal hæmoglobinuria the patient is often a child or young adult suffering from congenital syphilis The attack is excited by cold and may be induced by placing the extremities in cold water (Rosenbach test) There is a hæmolysin circulating in the blood, which at low temperatures attaches itself to the red cells and causes their lysis This is the basis of the Donath Landsteiner reaction Granular casts may be found in the urine in exercise hæmoglobinuria

**Treatment** This varies with the cause To avoid a mismatched blood transfusion adequate care must be taken in blood grouping Cold hæmoglobinuria can often be cured by appropriate anti syphilitic treatment In other cases daily intravenous injections of ascorbic acid mg 300 given for several days, have proved successful The outlook in the nocturnal hæmolytic anæmic group is very grave Repeated small blood transfusions should be given, and in no case should splenectomy be performed Exercise hæmoglobinuria is a condition somewhat analogous to postural albuminuria and usually disappears without special treatment

## Porphyrinuria

*(Porphyria)*

The term hæmatoporphyrinuria formerly employed, is incorrect Hæmatoporphyrin is not a naturally occurring substance Porphyrinuria may be due to drugs such as Trional or sulphonal or T N T poisoning or rarely it is a congenital metabolic error associated with photosensitivity skin lesions (hydroa æstivale) and pigmentation of bones and teeth An acute or intermittent idiopathic type is also described The symptoms are abdominal pain due to gastric or intestinal spasm vomiting and constipation peripheral neuritis with flaccid paralysis of the extremities, and the urine is brown or red The patient is often acutely ill and runs the risk of a laparotomy being performed if the significance of the urinary changes are not appreciated The urine may be of normal colour when passed darkening on standing to a brown or red shade (port wine colour) The pigment is detected spectroscopically Porphobilinogen the chromogen precursor of porphobilin is detected in the urine by the Ehrlich aldehyde test The red colour produced is distinguished from that resulting from the presence of urobilinogen as the porphobilinogen is insoluble in chloroform and ether

## Pneumaturia

Gas may be passed with the urine when there is a fistula between the bladder and bowel or an urethral fistula Occasionally it is due to the presence of gas forming organisms in the bladder

## Pyuria

**Definition** Pus in the urine

**Etiology** The pus may come from the kidney, as in pyelonephritis pyonephrosis, pyelitis or renal calculus from the ureter owing to the

presence of a calculus from the bladder in cystitis new growth a calculus or an adherent and perforated diverticulitis or appendix abscess and from the urethra in urethritis or in periurethral suppuration such as may result from a prostatic abscess The urine may be thick or the pus may be present only in microscopical amounts The best test for pus is the microscopical one of finding pus cells in the urinary deposit The treatment is that of the various causative conditions

### Renal Function Tests

In medical cases, in addition to a general clinical examination, these tests are important for three varieties of problems

1 Does albuminuria signify nephritis or permanent renal damage ?
2 What is the type and severity of nephritis in any given case ?
3 In cases of essential hypertension is there failure of renal function ?

The practitioner will require the following tests

✓*Examination of the Urine* The presence of protein casts and cells The determination of the relation of proteinuria to posture and exercise The amount of urine passed by day and by night and the specific gravity of the day and night specimens

*Examination of the Blood* An increase of urea and non protein nitrogen is associated with acute glomerulo nephritis and with malignant nephrosclerosis A considerable increase renders the prognosis more unfavourable suggesting the danger of uræmia

✓ *The Excreting and Concentrating Power of the Kidney* This can be roughly determined by the urea concentration test and the water concentration and elimination tests

*The Water Concentration Test* This is performed as follows The patient should have nothing to drink after lunch the day before the test The evening meal should be dry and consist mainly of protein The urine passed during the night is discarded After emptying the bladder at 7 a m and discarding the specimen the urine is collected at 8 a m 9 a m and 10 a m Normally the specific gravity of at least one specimen should be over 1 025

✓*The Water Elimination Test* The patient takes no food and no fluid after 8 p m The bladder is emptied at 7 a m and the specimen discarded 1 250 ml of water are then taken and the urine is passed and measured at 8 a m 9 a m 10 a m and 11 a m The total of the four specimens should normally be over 80% of the fluid taken The specific gravity of one specimen should be at least 1 003

*The Blood Urea Clearance Test (Van Slyke)* This shows the volume of blood whose urea content is excreted in one minute s urine With standard clearance ' the normal man excretes 1 ml urine a minute containing the urea content of 54 ml of blood The results can be expressed as a percentage figure *e g* the kidneys are found to be 60% efficient The normal figure is from 70% upwards

*The Injection of Dyes and their Excretion* These tests are only applicable if there is no blood in the urine and are chiefly of value in

determining the separate function of each kidney, the urine being collected by ureteric catheters

For practical clinical medicine the most simple and reliable tests are the examination of the urine for volume, specific gravity protein casts and cells, the blood chemistry the blood urea clearance and the urea concentration and water concentration and elimination tests In normal cases the urine is more concentrated by night the volume being less and the specific gravity being higher than in the day specimen The urine also contains no protein but a few hyaline casts and red corpuscles are of no significance

The normal blood chemistry figures are shown on p xx

## NEPHRITIS AND NEPHROSIS

### (*Bright's Disease*)

**Definition** Bright s disease includes inflammatory and degenerative changes of the kidneys Suppurative renal lesions are excluded *Varieties* The cause of the various types described is unknown and it is difficult to classify them in accord with both clinical and pathological findings The following is the classification adopted —

**Nephritis**

1 Acute glomerulonephritis
2 Focal nephritis
3 Acute interstitial nephritis
4 Subacute nephritis
5 Chronic glomerulonephritis
6 The kidney of pregnancy

**Arteriolar Nephrosclerosis**

**Nephrosis**

1 Lipoid nephrosis
2 Amyloid nephrosis
3 Chronic intercapillary glomerulosclerosis

**Acute Tubular Necrosis**

Ellis in 1942 proposed the following classification for Bright s disease —

| | |
|---|---|
| Nephritis | Type 1 nephritis<br>Type 2 nephritis |
| Essential hypertension | Benign hypertension<br>Malignant hypertension |
| Miscellaneous conditions | Acute focal nephritis<br>Toxæmia of pregnancy<br>Mercurial nephrosis<br>Amyloid nephrosis<br>Chronic pyelonephritis<br>Chronic nephritis of lead workers |

Type 1 nephritis corresponds with acute glomerulonephritis This has an abrupt onset Type 2 nephritis has an insidious onset The symptoms persist and recovery is unlikely Ellis includes many cases of nephrosis in this group Clinically it appears to correspond with cases described as chronic nephritis with œdema or chronic hydræmic nephritis The *histological changes are said to differ in the two* groups

This classification has not been widely adopted but is still included as it is used by teachers in certain medical schools in this country It is doubtful whether it has been received with enthusiasm by any pathologist or clinician with a wide experience of nephritis The reader cannot fail to note that in the Ellis classification the majority of renal conditions remain unclassified It appears that an attempt has been made to over simplify a difficult complicated and little understood subject Such attempts are likely to be misleading until the causes of the different varieties of nephritis or nephrosis are known Some pathologists have not been able to substantiate a characteristic renal difference between the two types of nephritis indeed in some cases which were clinically grouped as Type 1 pathological changes ascribed to Type 2 have been found The inclusion of nephrosis in Type 2 nephritis and the subsequent exclusion of lipoid nephrosis from this group appears illogical and lacking in foundation Clinically some cases which have been labelled as Type 2 on account of an insidious onset with œdema eventually lose the œdema and do not pursue the progressive course laid down for Type 2 nephritis

## Acute Glomerulonephritis

### (*Type* 1 *Nephritis*)

**Definition** Inflammation and degeneration affecting the glomeruli and tubules of the kidney

*Etiology* Acute glomerulonephritis is secondary to infection in some site in the body It is probably not directly due to organisms nor to their toxins but to a substance formed during the immunising process to which the kidney is sensitive The most common cause is the hæmolytic streptococcus associated with an upper respiratory tract infection the nephritic symptoms appearing 7 to 21 days after the onset of the infection It may be associated with tonsillitis dental infection cervical adenitis parotitis and subacute bacterial endo carditis The pneumococcus or streptococcus viridans may also be the causative organism Ellis found a history of preceding infection in 84% of cases It is met with also in association with Acute specific fevers especially scarlet fever (third week) and less frequently measles small pox, and diphtheria It may also occur in association with pregnancy and purpura It rarely follows an operation for the removal of septic tonsils a mastoid operation or antrotomy Certain chemical substances may produce a condition closely resembling acute glomerulonephritis These include mercury phosphorus arsenic cantharides sulphonamides carbon tetrachloride etc *Predisposing causes* 1 Age Children and

young adults 2 Sex Males predominate 3 Exposure to cold and alcohol which lower the resistance of the kidney

**Pathology** The kidney is normal in size or enlarged and the capsule strips readily In the early stages the kidney may be pale with ischæmic glomeruli or mottled with the glomeruli seen as red points or there may be increased vascularity the cortex being swollen and blood readily exuding Microscopically all the glomeruli are swollen and distended with shed endothelial cells blood cells and thrombi The tubules are affected after the initial glomerular changes the cells showing cloudy swelling and desquamation may occur Blood and casts may be seen in the tubules It is possible that the cutaneous capillaries are affected at the same time, accounting for the early appearance of the œdema Glomerular lesions are characterised by oliguria albuminuria and hæmaturia tubular lesions by polyuria the presence of casts and a low specific gravity urine

**Clinical Findings** Acute nephritis was very commonly met with *20 or 30 years ago, there were always a few cases in the wards of* general hospitals Today acute nephritis is very rarely seen indeed it is the exception to meet with any variety of nephritis in hospital This may be due to the effect of sulphonamides or penicillin in rapidly cutting short an attack of tonsillitis Clinically the disease begins in three ways conforming roughly to three types a hæmorrhagic, an exudative and a toxæmic or hypertensive type It may thus begin with hæmaturia micturition often being frequent and scanty with œdema, or with toxæmic symptoms such as sore throat dyspnœa headache backache limb pains nausea vomiting and diarrhœa or convulsions (hypertensive encephalopathy) Several of these symptoms *are often associated* and fever at the onset is common to each type

*On Examination* The patient looks slightly puffy or definitely swollen about the face he is generally pale and œdema may be present in the legs over the lumbar region (lumbar pad) over the sternum and in the scrotum The abdominal and *thoracic wall may* become œdematous and the swelling spread over the thighs After the swelling has subsided lineæ atrophicæ are sometimes observed Ascites or pleural effusion may develop later The swelling causes a *feeling of stiffness as if the body were in a leather case* and the patient often prefers to be propped up owing to a certain degree of dyspnœa There is usually fever at the onset with a temperature of 101 F to 103° F falling to normal in a week or 10 days During the acute stage the blood pressure is generally raised for a few days rarely above 160 or 180 mm Hg in an adult Cardio vascular system There is generally no cardiac enlargement which can be detected clinically or radiographically but the aortic second sound is often accentuated and a tic tac rhythm may be noted The pulse rate is increased The lungs A few scattered rhonchi or some basal rales are often heard There are usually no retinal changes but at times there are signs of hypertensive neuro retinopathy the arteries are narrowed veins dilated a few hæmorrhages are noticed or there may be slight œdema of the discs The subcutaneous œdema fluid is

rich in protein (over 1%) The urine The volume is reduced to about 10 to 20 oz or there may be anuria for a day or so. The specific gravity is raised (1 025 to 1 035) The colour is dark owing to blood and urates Protein is present (0 4 to 2%) Microscopically Hyaline blood and epithelial casts renal epithelial cells and some red and white blood cells are seen but the urine is usually sterile The blood Nitrogen retention often occurs during the acute stage The protein and cholesterol contents are normal The sedimentation rate of the red cells is raised The urea concentration test should not be performed owing to the strain it places on the kidney The water elimination test shows low values

**Differential Diagnosis** This usually presents little difficulty the signs and symptoms being characteristic Focal nephritis is excluded by the presence of œdema the hypertension and the blood nitrogen retention In some cases the disease is a reactivation of an old infection when cardiac hypertrophy and arteriosclerosis may be expected The presence of fatty casts in the urine is also suggestive of old standing disease Other causes of hæmaturia must be considered but these are not usually associated with œdema except in heart failure where the cardiac condition is obvious

**Course and Complications** Acute nephritis is often a self limited disease and about 80% of cases recover after a few days the urinary output suddenly increases to about 60 to 80 oz a day the amount of blood excreted diminishes and the blood pressure falls to normal The œdema may however persist for several weeks or the condition merge into one of subacute or chronic nephritis (Type 2 nephritis) Uræmia hypertensive encephalopathy myocardial failure pericarditis peritonitis and acute œdema of the lungs or larynx are of serious import Bronchitis is not usually severe

**Prognosis** Death is uncommon during the acute stage but may result from hypertensive encephalopathy uræmia left ventricular failure bronchopneumonia or streptococcal peritonitis In other cases death occurs within a few months Cases due to scarlet fever often make a complete recovery but there is always the danger of permanent renal damage

**Treatment** *Prophylactic* The use of penicillin and other antibiotics in the treatment of acute tonsillitis scarlet fever and other hæmolytic streptococcal infections is of great prophylactic value

*Curative* The patient should be in bed between blankets and in flannel pyjamas The temperature of the room should be maintained at 60 F to 65 F Diet The ordinary milk diet should not be given as this contains about 65 G of protein and is rich in salts The diet should be as follows *Diet* 1 Days 1 to 3 or 4 The total fluid should not be more than 1 pint in 24 hours made up of dextrose orangeade (dextrose 3 oz the juice of one orange and water 1 pint) water and barley water A little toffee and raw apple may also be taken When the urinary output increases and the hæmaturia diminishes *Diet* 2 is given This is milk ½ a pint diluted with water or soda water half and half Barley water or dextrose orangeade

1 pint Toffee Four thin slices of bread and butter One small cup of Benger's food Cream ½ oz Weak tea and sugar In another 3 or 4 days if the patient improves, the diet may be gradually increased by the addition of the following substances Milk up to 2 pints in all Bread and milk, sugar toast honey, potatoes and green vegetables Then add gradually milk pudding with no eggs, suet pudding porridge fruit such as bananas steamed fish chicken boiled mutton and an egg once a week *All meat and vegetable extracts which are rich in salts* and nitrogenous bodies must be avoided, also coffee alcohol, bacon, ham cheese, pickles, salt, and red meats No salt should be used in cooking or taken with food while any œdema is present This type of low protein diet is usually recommended but there is no proof that the course of the disease is affected by reducing the protein intake

The bowels should be opened daily with salts such as magnesium or sodium sulphate in doses sufficient to produce a loose motion (usually gr 60 to 120) If there is œdema, sweating may be induced by hot packs or by a hot air bath (using an electric cradle the temperature being raised to 120° F for 20 minutes) Care must be taken not to exhaust the patient, the pulse being watched all the time Hot applications should be placed over the loins to relieve aching Diuretics are best avoided but in cases of complete supression of urine an intravenous injection may be given of 250 ml of 0 9% sodium chloride solution containing 10% dextrose and 4 285% magnesium sulphate If there is evidence of an infective lesion in the throat or elsewhere a course of penicillin should be given The treatment of convulsive *(hypertensive) uræmia is pescribed on p 487*

*Convalescence* The patient must be kept in bed until all blood and casts have disappeared from the urine the œdema has gone and the temperature and sedimentation rate of the red cells are normal The associated anæmia is benefited by tab Fersolate gr 3 2 t i d p c The eradication of septic foci especially in teeth and tonsils is usually advised, but there is no definite evidence that tonsillectomy is of any *value in curing nephritis or preventing it from progressing* to a chronic stage

## Focal Nephritis

**Definition** A condition characterised by *hæmorrhages into certain* glomeruli of the kidney

**Etiology** Focal nephritis is due to bacterial emboli usually strepto coccal It occurs in association with such acute infections as tonsillitis otitis media erysipelas, puerperal fever pneumonia, scarlet fever (during the first week) influenza meningococcal meningitis etc In subacute bacterial endocarditis a condition of multiple glomerular *embolisation often occurs*

**Pathology** There is hæmorrhage into certain glomeruli and there may be cloudy swelling of the epithelium of the tubules In subacute infective endocarditis multiple infarcts occur in the glomerular vessels

**Clinical Findings** *The patient is a child or an adult who* during the acute stage of one of the illnesses mentioned above passes blood

in the urine The urine may be diminished in volume containing blood albumin and hyaline granular and blood casts Streptococci can often be found in the urine There is no œdema no increase of blood pressure and no nitrogen retention in the blood

**Differential Diagnosis** Focal nephritis is differentiated from acute diffuse glomerulonephritis by its onset during the acute stages of an infection and the absence of œdema and hypertension

**Course and Complications** The urinary changes usually rapidly disappear rarely albuminuria persists for some time

**Treatment** No special treatment is required for the renal lesion If there is oliguria the fluid intake should be restricted to 1 to 2 pints until diuresis ensues After recovery focal sepsis in the tonsils or teeth should be treated

### Acute Interstitial Nephritis

**Etiology** Acute interstitial nephritis occurs in association with diphtheria scarlet fever septicæmia and streptococcal tonsillitis

**Pathology** Areas of infiltration with red and white blood cells and plasma cells are seen in the cortex between the tubules and glomeruli

**Clinical Findings** It is impossible to diagnose this condition during life In some cases there is oliguria with slight albuminuria Usually there is no hæmaturia no œdema and no hypertension

### Subacute Nephritis

Subacute nephritis may occur as a stage in the course of acute glomerulonephritis the symptoms persisting for more than a few months from the onset In other cases it may be an exacerbation of chronic glomerulonephritis

### Chronic Glomerulonephritis

**Etiology** It is in this variety of nephritis that the greatest difficulty arises in classification Ellis differentiates between the variety of chronic nephritis which is a sequel of his Type 1 nephritis and that which arises insidiously (Type 2 nephritis) In the Type 1 the disease runs a prolonged course the patient often remaining in good health although albuminuria persists Eventually death occurs from hyper tension and renal failure In the Type 2 nephritis the onset is always insidious In 95% of cases there is no history of a related infection at the onset or of acute nephritis blood is rarely present in the urine and œdema is marked For clinical purposes we may recognise the type characterised by œdema (nephrotic nephritis) and that in which œdema is minimal but in which there is hypertension and renal failure In some patients the œdematous phase passes away before death occurs

### The Œdematous Type

**Clinical Findings** In the early stages the clinical picture closely resembles either that of subacute nephritis or of chronic nephrosis The characteristic features are the presence of œdema rise of blood

pressure, and retention of nitrogen in the blood The urine is either normal in amount or slightly diminished Sp Gr 1 020 to 1 026 Protein 0 5 to 1% Deposit epithelial cells, a few red and white blood cells and some hyaline and granular casts The urea concentration and water elimination tests usually indicate deficient renal function The œdema fluid is rich in protein over 1% In the nephrotic type the blood cholesterol may be raised and the plasma proteins diminished

**Differential Diagnosis** In some cases it is impossible to differentiate chronic glomerulonephritis from chronic nephrosis The history of previous acute nephritis a raised blood pressure cardiac enlargement retinal changes and the presence of more than a few red blood cells in the urine are characteristic of glomerulonephritis

**Course and Complications** The course is usually slowly progressive Exacerbations characterised by the presence of blood in the urine may occur The patient may die from renal failure or from intercurrent disease before a secondary contracted kidney develops The œdema may increase owing to the occurrence of nephrotic changes in the kidneys or of cardiac failure Complications also include pericarditis ulceration of the colon and bronchopneumonia

**Prognosis** This is very unfavourable in cases which do not respond to treatment and in which the œdema does not disappear It is also unfavourable if the disease progresses to the stage of secondary contracted kidney

**Treatment** The patient should be kept warm in bed during the œdematous stage A salt poor moderate protein diet containing about 60 to 80 G with a calorie value of about 2 200 should be given

*Moderate Protein Diet* **Breakfast** Egg 1 bread 3 oz or toast 2 oz butter ¼ oz milk 4 oz , marmalade or honey ½ oz tea 5 oz **Lunch** White fish (cooked) 3 oz potato 4 oz green vegetables 2 oz stewed fruit 2 oz butter ¼ oz bread 3 oz or toast 2 oz **Tea** Bread 2 oz butter ¼ oz jam ½ oz milk 2 oz tea 5 oz **Dinner** Chicken or lean meat (cooked) 3 oz potato 4 oz green vegetables 2 oz fruit 4 oz bread 2 oz butter ¼ oz Sugar for sweetening during day 2 oz

Bread made without salt should be used No salt must be added in cooking or subsequently The fluid intake should be restricted to about 35 oz according to the urinary output

*Drainage of Fluid* The peritoneal or pleural cavity may be aspirated or the legs drained by small incisions which are kept covered with sterile gauze while the patient sits up in a chair for 1 to 3 days

Massage is a useful adjunct to treatment especially when applied to the extremities For the anæmia which is usually present transfusion of packed red cells may be given Iron frequently has no effect in curing the anæmia The bowels should be kept open daily with fl oz ½ to 1 of Mist mag sulph alb (B P C)

## The Non œdematous Type

**Clinical Findings** The patient may complain of headache dyspnœa weakness frequency of micturition especially at night and symptoms

of chronic uræmia such as nausea vomiting or diarrhœa and loss of weight

*On Examination* The urine The volume is usually increased relatively more being passed by night Sp Gr 1 005 to 1 012 A trace of albumin may be present Deposit a few epithelial and blood cells and an occasional hyaline or granular cast The blood The urea and non protein nitrogen are raised the cholesterol and protein normal The heart There is a tendency to hypertrophy of the left ventricle and the blood pressure is raised The arteries are thickened Ophthalmoscopic examination Retinal hæmorrhages may be seen with tortuous arteries passing over constricted veins

In children the disease may occur insidiously and give rise to a condition of renal infantilism (also known as renal dwarfism or renal rickets) the growth being stunted the bones deformed and the secondary sexual characteristics in abeyance

**Differential Diagnosis** It may be impossible to differentiate this condition from essential hypertension

**Course and Complications** The course is progressive with gradual failure of renal function Complications due to hypertension such as convulsions or cerebral hæmorrhage may occur

**Prognosis** This is very unfavourable The patient usually dies from uræmia within 2 years of the appearance of retinal changes or of a marked rise in the blood nitrogen or death may be due to heart failure Pregnancy affects the condition adversely and should be prevented or terminated

**Treatment** This is as described for nephrosclerosis (see p 476)

## The Kidney of Pregnancy

The kidney of pregnancy must be differentiated from an exacerbation of chronic nephritis which when present usually shows itself during the first few months of pregnancy The kidney of pregnancy resembles a nephrosis in that the renal changes consist in a degeneration of tubular epithelium The cause is unknown It is especially liable to develop in the second half of pregnancy and in elderly primiparæ It differs clinically from a typical nephrosis in that the blood pressure often rises considerably and convulsive attacks (eclampsia) are liable to occur The renal function tests are usually normal including the blood urea and non protein nitrogen and the urea concentration test

## Arteriolar Nephrosclerosis

**Etiology** Arteriolar nephrosclerosis is a complication of hypertensive vascular disease

**Pathology** The kidneys may be normal in size slightly larger than normal or somewhat contracted Granulations are usually slight The colour may be dappled by red and yellow areas The changes in the arterioles are secondary to hypertension There is hyaline degeneration beneath the endothelium of the arterioles which narrows their lumen followed by atrophy of muscle fibres and replacement fibrosis In

addition to the changes in the arterioles and degeneration of the glomeruli and tubules, hæmorrhages may be seen

**Clinical Findings** The patient is usually an adult between the ages of 30 and 40 and more often a male The condition may occur in children The onset is often insidious but in others it is sudden with a cerebral hæmorrhage or uræmic attacks

*On Examination* The heart is hypertrophied and the arteries thickened The blood pressure is high, the systolic being usually over 200 mm Hg and the diastolic between 120 and 160 mm Hg In the "benign type" of the disease there is no evidence of renal failure and no renal retinopathy Later, however the malignant phase develops *Ophthalmoscopic examination* Hypertensive neuro retinopathy is usually present The disc is swollen red and its edges blurred The retina shows fluffy white spots ("cotton wool" areas) due to exudate and fatty changes *Hæmorrhages are present* The arteries are narrowed ("silver wire" appearance) and the veins dilated The urine The volume may be as much as 80 or 100 oz with an increase in the nocturnal output In the later stages the excretion falls and a fixed amount tends to be passed every hour by day and night Sp Gr 1 002 to 1 008 Protein a trace to 0 5% Deposit occasional renal epithelial and red cells and hyaline and granular casts The blood Urea and non protein nitrogen are increased to 100 mg per 100 ml or more Cholesterol normal Creatinine may be raised to 2 5 mg per 100 ml Calcium may be low 6 mg per 100 ml or less The urea concentration and water elimination tests show low figures as the disease progresses

**Differential Diagnosis** The differentiation from chronic glomerulo nephritis can only be made if the patient is seen in the early stages The heart changes and high blood pressure precede the albuminuria and cylindruria in chronic glomerulonephritis

**Course and Complications** The course is slowly progressive a cerebral hæmorrhage uræmia heart failure and secondary infections such as bronchopneumonia are liable to occur Terminal pericarditis and pleurisy are probably toxic in origin Acute œdema of the lungs is rare but attacks of nocturnal dyspnœa (renal asthma) with Cheyne Stokes breathing may occur Exacerbations of an acute nephritic type may also be noted with œdema increase of the albumin and the presence of more red cells in the urine

**Prognosis** This is always very grave although the patient may live for several years Unfavourable signs are a blood urea figure of over 200 mg per 100 ml and the presence of hypertensive retinopathy In 90% of cases death occurs within 2 years of the diagnosis of the latter

**Treatment** The general regime consists in regular hours moderation in work exercise diet alcohol and smoking The two latter are best avoided completely The most suitable climate for the winter is that of Egypt but if it is not possible to go abroad chills should be avoided Diet The protein *allowed is determined to a certain degree* by the blood nitrogen figures The basal requirement is about 0 5 G of protein for every 1 lb of body weight or 70 G for a man of 10 stones

With a blood urea of about 60 mg per 100 ml the patient can be put on the moderate protein diet (see p 474)

With a blood nitrogen of over 100 mg per 100 ml a low protein diet containing about 35 to 40 G of protein and a calorie value of 1 700 can be given for periods of 2 or 3 weeks alternating with the moderate protein diet

*Low Protein Diet* **Breakfast** Oatmeal (uncooked) 1 oz bread 2 oz butter ½ oz milk 4 oz tea 4 oz **Lunch** Green vegetables 2 oz potato 4 oz bread 2 oz butter ½ oz stewed fruit 2 oz cream ½ oz jam ½ oz rice (uncooked) ½ oz **Tea** Bread 1 oz butter ½ oz tea 4 oz jam ½ oz milk 2 oz **Dinner** Vegetable soup white fish or lean meat or chicken (cooked) 2 oz potato 4 oz fruit 4 oz bread 1 oz Sugar for sweetening during the day 2 oz

The bowels should be kept acting freely with the help of salts (mag sulph or sod sulph gr 60 to 120) in the morning For insomnia chloral hydrate gr 5 and sod brom gr 10 may be given at night For headache with high blood pressure tabella glyceryl trinitrat gr 1/130 may be given 3 times a day but it is not wise to endeavour to lower the blood pressure considerably For dyspnœa due to acidosis alkalis such as sod bicarb gr 20 may be given six hourly and if there is heart failure with œdema digitalis should be given The treatment of uræmia is considered later

## THE NEPHROSES

### Lipoid Nephrosis

**Etiology** The cause of lipoid nephrosis is usually unknown It may be due to a disturbance of lipoid metabolism

**Pathology** It is believed that there is increased glomerular permeability to protein and lipoids The smaller albumin molecules pass through rather than the larger globulin one The function of the tubules is not impaired as shown by the high Sp Gr and ammonia content of the urine The absence of a high blood pressure indicates that the renal circulation is not affected The kidneys are either normal in size or enlarged The capsule strips readily the cortex is enlarged and shows yellow streaks Microscopically there is lipoidosis of the tubular epithelium

**Clinical Findings** The nephrotic syndrome is characterised by massive œdema heavy albuminuria hypoproteinæmia elevation of blood lipoids and cholesterol and depression of the basal metabolic rate The patient is usually a child or young adult The disease begins insidiously with languor headache pallor swelling of the face legs scrotum or abdomen and possibly some nausea or diarrhœa

*On Examination* There is a fairly generalised œdema and ascites and hydrothorax may also be present The face is pale but often there is no real anæmia and no cardio vascular changes develop The blood pressure is not raised Retinal changes do not occur The œdema fluid contains little protein less than 0 1% The basal metabolic rate

is low The urine Volume 20 to 30 oz Sp Gr 1 020 to 1 040 Acid (pH 5 4) Protein 1 to 6%, chiefly albumin There may be a trace of sugar Deposit, leucocytes and epithelial cells and occasionally a red cell Hyaline epithelial granular and fatty casts and doubly refractile lipoid particles Urea concentration test normal Water elimination test shows low excretion This is due to pre renal deviation and not to failure of renal function The blood Protein about 4% (normal being 7 4%) of which 89% is globulin (normal being 37% globulin) There is retention of cholesterol (300 to 2 300 mg per 100 ml, the normal being 140 to 280 mg per 100 ml), and lipæmia The urea and non protein nitrogen are normal

Such a case as this conforms to the type described as lipoid or true" nephrosis the renal changes being degenerative rather than inflammatory and it is differentiated from the nephrotic type of chronic glomerulonephritis by the history and the tendency to hypertension in the latter and from amyloid nephrosis in which there are associated suppurative syphilitic or tuberculous lesions

**Course and Complications** Complete recovery is said to be the rule if proper treatment is given but this is not always the case The course is variable and death may result either from failure of renal function with gradual increase of blood nitrogen and eventually uræmia or from complications such as pneumococcal peritonitis and erysipelas

**Treatment** The Wassermann reaction should be determined and if positive a course of penicillin given (see p 596) It is not now considered necessary to give the high protein diet recommended by Epstein For an adult a diet may be given containing protein 100 to 130 G carbohydrate 250 G and fat 80 G A child requires protein 2 3 G per kg bodyweight and enough fat and carbohydrate to yield a high calorie diet Fluid should be restricted to 40 to 45 oz No salt must be used in cooking or added to the food

A preliminary venesection followed by a blood transfusion assists in restoring the blood proteins to their normal value

Thyroideum may also be given beginning with gr 1 t d s and working up to large doses such as gr 15 to 40 daily as long as the blood cholesterol is raised This is especially indicated in cases with a low metabolic rate

Mersalyl may be administered cautiously together with ammon chlorid (see p 241) if there is no nitrogen retention The legs abdomen and thorax may also be drained if necessary

Other treatments recommended include the intravenous injection of dextran the administration of cation exchange resins of ACTH or cortisone and malaria therapy

Dextran is injected intravenously to increase the colloidal osmotic pressure of the blood Dextraven is a 10% salt free solution of clinical dextran 540 ml are injected intravenously at intervals of about 3 days until the œdema is considerably reduced

The cation exchange resins consist of porous molecules arranged in a lattice pattern Katonium is a sulphonated polysterene resin with

75% of the cation in the $NH_4$ phase and 25% in the K phase In the small intestine the resin exchanges its $NH_4$ and part of its K for Na ions In the colon some of the Na ions return to the intestinal contents in exchange for K The net result however is a gain of K and Na ions by the resin in exchange for $NH_4$ ions The latter are absorbed converted into ammonia and hydrogen and acidosis ensues The healthy kidney then excretes a highly acid urine and there is an increased elimination of Na in the urine and fæces especially if the motions are kept loose Excessive loss of K in the stools may lead to hyperchloræmic acidosis and hypopotassæmia The treatment must therefore always be carefully controlled by electrolytic determinations

Katonium is a powder which is given in doses of 15 G q i d in fluids such as fruit juices between meals Prolonged courses consist of 6 days of the powder and then 2 days without and so on During the 2 day periods in which no resin is taken potassium citrate gr 15 is given t i d The treatment may be continued for weeks or months in suitable cases In addition the patient must be given a high protein low salt diet Good results have been obtained by this means in some cases of the nephrotic syndrome as well as in cardiac œdema

### Amyloid Nephrosis

**Etiology** Amyloid nephrosis may occur in connection with chronic pulmonary tuberculosis syphilis or chronic suppuration

**Pathology** The kidney is enlarged smooth and pale The capsule strips readily The glomeruli show on section as dark brown spots when stained with iodine Later atrophy and fibrosis result in a small firm contracted kidney Microscopically it is seen that amyloid is deposited around the capillaries and in the walls of the arterioles and venules. The glomerular vessels may be the only ones affected Fatty and atrophic changes occur in the cells of the first convoluted tubules

**Clinical Findings** Œdema develops and the urine often contains a considerable amount of albumin There may be polyuria The blood changes resemble those found in lipoid nephrosis The total protein may be as low as 4% due chiefly to loss of albumin The blood cholesterol is raised 300 to 600 mg per 100 ml The nitrogen figures are normal except in cases of amyloid contracted kidney in which there is azotæmia

**Treatment** No special measures are indicated for the nephrosis the underlying cause being the essential factor for treatment Any septic focus should be removed A sufficiency of protein should be given in the diet to compensate for the protein lost in the urine

### Chronic Intercapillary Glomerulosclerosis

*(Kimmelstiel Wilson Kidney)*

**Pathology** There are probably two types of intercapillary glomerulo sclerosis a diffuse one with widespread thickening of Bowman s basement membrane and a nodular found in diabetes and called the Kimmelstiel Wilson lesion in which there are islands of hyaline material in the

glomeruli There is also thickening of Bowman's membrane to form a hyaline cap and arteriolar sclerosis of the afferent and efferent glomerular arterioles

**Clinical Findings** The characteristic features are diabetes mellitus with heavy albuminuria œdema, retinopathy peripheral neuropathy and cardiac failure with hypertension Elderly women are chiefly affected Hypoproteinæmia may occur in some cases

## Acute Tubular Necrosis

**Definition** A condition characterised by acute degeneration affecting especially the ascending loops of Henle and the distal tubules

**Etiology** The lesions are due to various poisons or toxic substances, or to ischæmia 1 *Bacterial toxins* The so called febrile albuminuria The renal damage is usually slight 2 *Chemical substances* Mercury bismuth arsenic phosphorus, carbolic acid cantharides and sulphonamides 3 *Metabolic products* Acidosis in diabetes mellitus Metabolites which should be detoxicated by the liver have to be dealt with by the kidney when the liver function fails This may occur in liver disease or after operations on the biliary tract 4 *Hæmoglobinuria* This may result from various causes such as a mismatched blood transfusion paroxysmal hæmoglobinuria blackwater fever mushroom poisoning and the crush syndrome 5 *Shock and low blood pressure* These may be due to injury hæmorrhage concealed accidental hæmorrhage an operation abortion coronary thrombosis cholera or high intestinal or pyloric obstruction

**Pathology** In severe cases there is a precipitation of casts in the ascending portions of the loops of Henle with tubular epithelial necrosis The glomeruli may be swollen with an exudate in the intercapsular spaces Cortical ischæmia may occur in the crush syndrome due possibly to a reflex cortical vascular spasm

**Clinical Findings** The clinical findings are not those of the nephrotic syndrome The characteristic feature of tubular necrosis is suppression of urine or anuria This results in an altered water balance and a retention of metabolites Potassium is liable to accumulate in the extracellular fluid which leads to muscular paralysis and cardiac failure This is particularly common in the crush syndrome where potassium is liberated from the damaged muscles

## The Crush or Compression Syndrome

**Etiology** This syndrome which was described in Germany in the 1914–18 war attracted attention in England as the result of injuries sustained in air raids in the 1939–45 war It is liable to occur when a limb is crushed by fallen masonry etc Accidents in civil life such as a fractured pelvis with central dislocation of the hip causing obstruction of the external iliac vessels may result in the syndrome without there being apparent muscle damage It has also been described after a difficult labour

**Clinical Findings** The patient on admission to hospital may or may not be suffering from shock In the former case he is pale sweating

and clammy the blood pressure is low the pulse feeble and the Hb % increased The affected limb is swollen and œdematous there may be areas of cutaneous erythema and vesicle formation Pulsation may or may not be present in the peripheral arteries The muscles feel firm the power of moving the limb is restricted and paræsthesia may be present Signs of renal failure are noted about the first or second day Early signs of failure are oliguria or even anuria and the appearance of blood and albumin in the urine Brown casts of epithelial cells are present and the pigment is shown spectroscopically to be myohæmoglobin The urinary urea and chloride figures fall the blood urea and potassium rise and the alkali reserve falls The blood pressure tends to rise and remain above normal if the patient is transfused with serum or plasma The terminal uræmic stages comprise vomiting excessive thirst coma and death When the serum potassium rises above 7 or 8 mEq /L (normal 4 4 to 5 6) the electrocardiogram shows changes The T waves are tall and spiked the QRS complexes are broad the P wave is absent and various arrhythmias develop When the potassium figure reaches 8 to 12 mEq /L flaccid muscular pareses are liable to develop It thus appears that both a rise and a fall in blood potassium results in muscular weakness due perhaps to transference of potassium across the cell membrane

**Prognosis** Recovery occurs in about one third of the cases The length of time that the limb has been compressed appears to bear no direct relationship to the prognosis A fall in the blood urea is a favourable sign In fatal cases death usually occurs between the fifth and eighth days

**Treatment** Anuria or oliguria may be treated by Borst s method There is a risk of inhalation of the oily material if it is vomited A continuous intragastric drip is given the fluid containing dextrose 400 G peanut oil 100 G muc acac q s and water to 1 litre All vomit is collected filtered through lint and returned to the stomach through the drip The total fluid given in 24 hours is 1 litre plus the volume of urine passed in the previous 24 hours Sodium and potassium are balanced quantitatively by daily observations on the serum contents When the urine output exceeds 1 litre a day a low protein diet is given instead of the drip feed If the hæmoglobin is below 70% a transfusion of packed red cells is given The best way of dealing with the compressed limb is a matter of dispute Some advise that a tourniquet should be applied as soon as the weight is removed from the limb and that decompression should subsequently be slowly effected when a good outflow of urine has been obtained Others recommend that the limb should be packed in ice or that incisions should be made to release the œdema fluid Primary amputation of the limb with the idea of saving the kidneys has resulted in the loss of both life and limb The alternative view based on the theory that the blood has lost vital principles into the œdema fluid is that the compression should be rapidly relieved the limb bandaged from below upwards and intermittent pressure up to 60 mm $H_g$ applied over the œdematous limb using a specially large sphygmomanometer cuff

### Other Causes of the Nephrotic Syndrome

These include thrombosis of the renal veins and poisoning with drugs such as troxidone (Tridione)

### The Pathogenesis of Renal Œdema

*Acute Glomerulonephritis* The œdema here cannot be due to protein loss in the urine The plasma proteins are normal and the œdema fluid *is rich in protein 0 6 to 1% The factors involved include* oliguria due to glomerular damage, and increased reabsorption of sodium and water from the renal tubules It is probable that the cutaneous capillaries are simultaneously damaged with those of the kidneys A further factor is congestive heart failure with rise in the capillary blood pressure

*The Nephrotic Syndrome* The following factors are probably concerned *with the production of œdema* —

1 **Hypoproteinæmia** The colloid osmotic pressure is lowered by loss of protein in the urine and hence fluid passes from the blood into the tissue spaces The normal colloid osmotic pressure is between 40 and 50 cm water in nephrosis the figures vary between 5 and 20 cm water The protein lost from the plasma is albumin rather than globulin owing to the smaller size of its molecule The protein content of the plasma in nephrosis does not usually fall below 4 G % A further fall in the albumin figure is compensated by a rise in the globulin content A second factor which may be partially responsible for the fall of the albumin content of the plasma is failure to synthesise new protein from amino acids The capillary permeability does not appear to be increased in nephrosis as the œdema fluid and ascitic fluid protein content is low

2 **Sodium Chloride Retention** This is of great importance It has been shown experimentally that if there is no sodium in the diet the plasma proteins may be low without the production of œdema It is not however believed to be the chief cause of œdema in nephrosis and *it is suggested that sodium chloride is retained in the* body in nephrosis in order to maintain a nearly physiological amount of sodium chloride in the tissue fluids and plasma If this is so the tubules must have the power despite the disease of the kidney of reabsorbing sodium chloride

3 **Reduction in Plasma Volume** Hypoalbuminæmia cannot be the only cause of œdema because diuresis may occur in nephrosis without any rise in the albumin content of the plasma This diuresis may be due to an increase in plasma volume

## URÆMIA

**Definition** Toxæmia due to or associated with failure of renal function

**Etiology** The cause is uncertain and it probably differs in the various types of uræmia A distinction may be drawn between prerenal renal and postrenal causes of uræmia

*Prerenal Causes* The primary changes occur in tissue metabolism in the chemical composition of the blood and in the circulation There is probably secondary renal insufficiency although no structural changes may be demonstrable in the kidneys The causes may be listed as follows Vomiting due to pyloric and intestinal obstruction and hyperemesis gravidarum Diarrhœa especially infantile and that due to cholera Hepatic disturbances as after operations on the gall bladder and bile ducts and cholæmia associated with acute hepatosis and cirrhosis Diabetes mellitus with acidosis especially in untreated cases Addison s disease during a crisis Severe hæmorrhage particularly hæmatemesis Post operative and traumatic shock Severe burns Peripheral circulatory collapse as in diphtheria and pneumonia Coronary thrombosis associated with shock Overdosage with alkalis in the treatment of peptic ulcer A mismatched blood transfusion and the crush syndrome

*Renal Causes* These include inflammatory degenerative and developmental changes in the kidneys such as acute and chronic nephritis bilateral renal suppuration acute tubular necrosis and congenital cystic disease

*Postrenal Causes* There is obstruction to the outflow of urine This may be due to mechanical causes such as calculi blocking both ureters a calculus obstructing the sole functioning kidney removal of the only existing or functioning kidney accidental ligature of both ureters in a pelvic operation pressure of a pelvic carcinoma on both ureters or a vesical growth obstructing the orifices of both ureters Incomplete obstruction may result from an enlarged prostate a pelvic tumour or a bilateral hydro or pyonephrosis Reflex inhibition of urinary secretion may be caused by a drainage tube placed in the bladder after prostatectomy

**Pathogenesis** There is always some retention of urea and other non protein nitrogenous substances in uræmia These are not considered to be the cause of the uræmic symptoms There may be a high blood urea content without uræmia The most important causes of uræmia are disturbances of electrolytic and water balance The various factors which may be involved are —

1 *Acidæmia* Normally the kidney forms ammonia to neutralise acids which are excreted Further by the excretion of more acid sodium phosphate than alkaline sodium phosphate acid excretion is increased If the kidney fails to manufacture ammonia and to excrete phosphate and sulphate acidæmia results This may lead to a low blood bicarbonate figure with clinical manifestations of acidosis such as hyperpnœa

2 *Alkalæmia* This is uncommon in uræmia It usually results from vomiting but may be due to a high alkali intake Alkalosis is characterised by headache dislike of sweet foods and tetany

3 *Disturbance of Water Balance* Polyuria with excessive sodium loss results in dehydration The anorexia and vomiting which are often present in uræmia increase the dehydration

4 *Hyperpotassæmia* The potassium figure in the blood may rise

to over 7 mEq/L (normal 4 45 to 5 6 mEq/L) This may result in sudden death from cardiac failure The electrocardiogram shows the changes described on p 481

5 *Hypopotassæmia* The characteristic features are flaccid paralysis The electrocardiogram shows small, flat or inverted T waves with prolongation of the Q–T interval

6 *Hypocalcæmia* The serum calcium is often low in uræmia This may be due to increased phosphate in the blood Twitchings or tetany may ensue

In many of the conditions such as hæmorrhage and shock, there is a fall in blood pressure which reduces the glomerular filtrate, and also renal anoxia which causes damage to the renal epithelium In Addison s disease there is further an alteration in the electrolytic balance of the blood and a rise in the hydrogen ion concentration Excessive vomiting may produce azotæmia before alkalosis occurs owing probably to loss of chlorides In alkalosis due to excessive intake of alkalis there is presumably some renal impairment In a mismatched blood transfusion and in the crush syndrome renal failure results from blockage of tubules with epithelial and blood pigment *débris* and from damage to tubular epithelium and cortical ischæmia

## Acute Uræmia

This is associated with certain types of acute hæmoglobinuric nephrosis such as result from a mismatched blood transfusion or crush injuries to a limb It is a true uræmia there is azotæmia and it is prerenal in origin The uræmic symptoms quickly follow on the exciting cause and the course of the syndrome whether for better or for worse is of short duration Treatment may be given by the Borst regime (see p 481) or 1 litre of 40% dextrose solution is given daily by intra gastric drip If there is vomiting the dextrose solution may be injected through a polythene catheter into the saphenous vein or into an antecubital vein In some cases peritoneal dialysis has proved successful Twice normal saline solution is given intraperitoneally by drip injection the fluid being later drained off The urea is eliminated into the peritoneal fluid The artificial kidney ' is sometimes employed

## Chronic Uræmia

**Clinical Findings** It is usually known that the patient is suffering from chronic nephritis The onset of uræmia is generally insidious and the symptoms may be grouped under the headings of the gastro intestinal nervous and respiratory systems *Gastro intestinal symptoms* There is thirst an unpleasant taste which may be bitter or ammoniacal due to excess of urea and ammonia in the saliva and loss of appetite There may be nausea vomiting diarrhœa associated with uræmic ulceration of the colon or constipation *Nervous symptoms* The patient may complain of general weakness inability to concentrate and insomnia Persistent dull headache is often an early symptom in addition to which muscular twitchings may occur Later there may be

hiccough convulsions amaurosis and finally coma *Respiratory symptoms* Various types of dyspnœa are described such as nocturnal attacks (renal asthma) Cheyne Stokes breathing or air hunger The hissing breathing is not often heard and is usually produced in the nose *Cardiac symptoms* Cardiac failure and terminal pericarditis may occur The skin is often dry of a yellow tinge and pruritus is trouble some A skin frost (urea crystals) is rarely seen Crystalline deposits on the skin may be due to sodium chloride Some of the deposit should be scraped off the skin and treated on a glass slide with a few drops of dilute nitric acid After evaporation if the frost is due to urea crystals of urea nitrate will be seen The temperature is usually subnormal and the patient is emaciated and dehydrated The blood and cerebro spinal fluid show a rise in urea and non protein nitrogen

**Differential Diagnosis** If the non protein nitrogen of the blood is low in patients presenting the signs of chronic nephritis it can usually be assumed that chronic uræmia is not present Alkalosis can be confirmed by determining the alkali reserve of the blood

**Prognosis** This depends upon the cause which sometimes can be removed as in the case of an enlarged prostate Some of the conditions such as alkalosis due to overdosage with alkalis respond rapidly to treatment (see p 669) In others such as malignant renal arterio sclerosis the outlook is hopeless Sudden death may occur in a patient who appears comparatively well

**Treatment** This must be directed primarily to the causative condition and secondarily to the relief of symptoms and the restoration of the electrolytic and water balance as far as possible Circulatory renal inefficiency which is a factor in many cases of prerenal uræmia must be treated and postrenal obstruction removed

The patient should be given a low protein diet (see p 477) for 3 or 4 weeks which can then be alternated with a moderate protein diet A low protein diet is advisable because it is possible that certain of the protein metabolites are toxic Further it reduces the amount of urea which has to be excreted and as urea causes osmotic diuresis this disturbs the water and electrolytic balance Many protein substances also contain potassium which is a toxic substance in excess in uræmia Treatment by the artificial kidney or by peritoneal lavage is usually not advisable

Various remedies have been recommended for the nausea and vomiting including Largactil mg 25 t i d or 500 ml of 1/6 molar sodium lactate solution intravenously In severe vomiting nutrition can be maintained for several weeks by infusions through a polythene tube inserted into an antecubital vein and passed into the superior vena cava Dextrose in strengths of 30% to 50% can be transfused through this tube Insomnia and headache may be relieved by Nembutal gr 1½ capsule once or twice daily For hiccough inhalation of oxygen may be temporarily effective Severe diarrhœa should be treated by the oral administration of streptomycin 1 G b i d Aperients should not be given owing to the risk of provoking uncontrollable

diarrhœa Anæmia requires transfusion of whole blood or of packed red cells

Sodium chloride solution must be injected intravenously to compensate for extracellular dehydration The amount of fluid required can be determined by estimation of the specific gravity of the plasma For every 0 001 rise in the Sp Gr of the plasma above 1 027 200 ml of water are required Sodium deficiency sometimes occurs without dehydration and this results in cellular overhydration The amount of sodium required is determined according to the information given in Chapter VI For the maintenance of fluid balance a daily intake of 800 to 1 000 ml plus a volume of fluid equal to the previous day s volume of urine is given

For the correction of acidosis sodium bicarbonate should be given by mouth in doses of 2 to 6 G or more according to the plasma $HCO_3$ level Alternatively isotonic sodium lactate can be administered intravenously

For calcium deficiency an injection of 10 to 20 ml of 5% calcium chloride solution should be given intravenously

Hyperpotassæmia can be remedied by giving dextrose by mouth in doses up to 200 G daily covered by an injection of insulin Hypopotassæmia requires the administration of potassium by mouth or intravenously (see p 506)

In difficult cases an accurate estimation of the fluid and electrolytic intake and output together with the level of the serum electrolytes may be necessary for the correct adjustment of fluid and electrolytic administration

### Latent Uræmia

*(Urinæmia)*

This occurs when there is complete failure of renal function and is due to postrenal mechanical causes (see p 483) The symptoms are entirely due to retention of urinary substances there is no element of dehydration or of hypertensive encephalopathy Beyond anuria there may be no symptoms noted for the first 4 or 5 days The patient then becomes a little drowsy the temperature falls and the pupils contract *The blood pressure does not rise* The blood urea and non protein nitrogen figures are increased to over 200 mg per 100 ml, and the alkali reserve falls to about 30 ml $CO_2$% Vomiting and muscular twitchings may usher in the final stage of coma Death usually occurs in from 10 to 14 days Unless the obstruction such as a calculus can be removed no treatment is available

### Acute Convulsive, Epileptiform or Eclamptic Uræmia

*(False Uræmia Hypertensive Encephalopathy)*

**Etiology** This is due either to acute glomerulonephritis or to hypertensive vascular disease

**Pathology** In acute glomerulonephritis there is probably cerebral œdema in hypertensive vascular disease cerebral vascular spasm

**Clinical Findings** The patient is usually suffering from acute glomerulonephritis but acute symptoms may occur in association with chronic nephritis or in cases of hypertension in which there is no nitrogen retention in the blood It does not occur in association with nephrosis as the blood pressure is not then raised The blood pressure rises and the patient is suddenly seized with convulsions or becomes comatose The convulsions may be local or general and consist of a tonic and clonic stage which may or may not be followed by coma Amaurosis of one or both eyes may also occur On ophthalmoscopic examination spasm of the retinal vessels may be seen or changes characteristic of hypertensive neuro retinopathy (see p 476) In some cases there is a prodromal stage characterised by anorexia headache and paræsthesia (see also p 281)

**Course and Complications** The convulsions may rapidly cease or prove fatal In some cases there is an ensuing period of coma

**Differential Diagnosis** Coma and convulsions are symptoms of widely different pathological states The diagnosis is usually established by the history of the case as regards renal disease the exclusion of other signs of organic nervous diseases and the examination of the urine blood cerebro spinal fluid blood pressure and discs In lead encephalo pathy in addition to the convulsions there is often retention of blood nitrogen possibly due to renal vascular spasm

**Prognosis** This is always grave but it is most favourable in cases of acute nephritis

**Treatment** The patient should be placed lying down dentures removed and care taken that an airway is maintained If he can swallow mag sulph gr 60 in water is given by mouth and repeated in an hour An intravenous injection of 40 ml of 30% sodium chloride solution is then given the patient being quietened during the injection by inhalations of chloroform if necessary If these measures fail venesection or lumbar puncture may be performed to lower the blood pressure and intracranial tension If a lumbar puncture is made the foot of the bed should be raised as there is a danger of the medulla being impacted in the foramen magnum and the fluid should be removed slowly Further depletive measures include an enema at night followed next morning by the rectal injection of 8 oz of water containing 2 oz of mag sulph (a 25% solution) run in slowly as for a colonic washout and retained as long as possible usually ½ to 2 hours The skin should be encouraged to act by a hot air bath using an electric cradle and raising the temperature of the bed to 120 F for 10 to 15 minutes The bowels should subsequently be kept well opened daily with mag sulph gr 60 to 120 mane and plenty of fluids should be taken by mouth

## BACTERIAL INFECTIONS OF THE RENAL TRACT

### Bacilluria

**Definition.** The presence of bacteria in the urine not accompanied by local symptoms

**Etiology** The organisms may gain access to the urine from the blood, being excreted by the kidneys from the intestines through the lymphatics in connection with the kidneys or by an ascending infection of the urethra Excretion of organisms by the kidneys implies a certain degree of renal damage Such organisms as the Bact *coli*, the *Proteus* vulgaris (B proteus), the Pseudomonas pyocyaneus the B typhosus the B paratyphosus A, B or C the Mycobacterium tuberculosis the *streptococcus the staphylococcus*, the Neisseria gonorrhœæ (gonococcus) or the Diplococcus pneumoniæ (pneumococcus) may be found Chronic constipation is not a predisposing cause In women habitual looseness of the bowels may be of etiological significance

**Clinical Findings** The history is of value Thus the bacilluria may be a sequel of an acute illness, such as typhoid fever or occur during an acute illness such as pneumonia bacterial endocarditis or gonorrhœa It may also be noted in cases whose chief symptom is debility, as in certain Bact coli infections or there may have been no symptoms attributable to the bacilluria

**Treatment** This depends upon the associated conditions for which the *special sections should be consulted*

**Bact coli Infections of the Urinary Tract**

**Pathology** The Bact coli may gain access to the urinary tract by three routes By the blood from the gall bladder or intestine by lymphatics from the intestine or by ascending infections of the urethra to the bladder and thence by periureteral lymphatics to the renal pelvis The lesions vary from slight degrees of cystitis and pyelitis to definite pus formation in the bladder or kidney

**Clinical Findings** The symptoms occur insidiously or have an acute onset, and are met with in children or in adults rather more frequently in women than in men Cases of Bact coli infection can be grouped clinically as follows 1 *Children* A cause of unexplained pyrexia and rigors 2 *Women* Before and after childbirth 3 *Adults* Cases fall into the following groups (*a*) There may be no symptoms Bact coli being found in the urine on routine examination (*b*) Acute renal or vesical symptoms such as pain in the loin or in the hypo gastrium *hæmaturia frequent, painful and scanty micturition* (*c*) Acute abdominal symptoms Acute pyelitis may be mistaken for appendicitis or salpingitis (*d*) General constitutional symptoms There is malaise and a continuous pyrexia *The condition is liable to be confused with* paratyphoid fever or other causes of obscure pyrexia In children it may be mistaken for tuberculosis (*e*) Chronic or recurrent *renal or* vesical symptoms There may be aching *in the loin or back* with frequency of micturition and feverish attacks

The urine This is acid and contains Bact coli a few *pus cells and* renal epithelial or vesical cells With cystitis there may be larger quantities of pus At the onset of pyelitis there may be a definite attack of hæmaturia The odour of the urine is often ' fishy and the organisms impart to the urine a characteristic sheen *A trace of albumin* is usually present

**Differential Diagnosis** The diagnosis is established by the urinary examination a catheter specimen is usually required to exclude the possibility of contamination Ureteric catheterisation will show which kidney is affected in cases of cystitis secondary to pyelitis In persistent cases X ray examination and pyelography should be made to exclude the presence of calculus or other lesions

**Course and Complications** If untreated the organisms usually persist in the urine and there is a danger of further infection occurring such as pyelonephritis and pyonephrosis

**Prognosis** This is very favourable in cases which are recognised and treated early If neglected it is more difficult to render the urine sterile

**Treatment** This is considered under the section dealing with pyelitis and cystitis (see below)

## Pyelitis

**Definition** Inflammation of the pelvis of the kidney

**Etiology** Pyelitis is usually due to infection with the Bact coli as described above Pyelitis may also be associated with a renal calculus renal tuberculosis obstruction of the ureters due to a calculus abdominal tumour or some anatomical abnormality such as bifid pelvis double ureter nephroptosis congenital hydronephrosis It may be associated with cystitis or urethral obstruction

*Varieties* Pyelitis may be acute or chronic

### Acute Pyelitis

**Clinical Findings** The patient who is suddenly taken ill complains of malaise due to the fever and toxæmia There may be no other symptoms or he may complain of aching or tenderness in the loin with frequency of micturition There may also be rigors

*On Examination* The temperature is found to be raised and there is usually abdominal tenderness over the affected kidney The urine shows the changes described above

**Differential Diagnosis** Acute pyelitis is liable to be mistaken for appendicitis salpingitis or if occurring after childbirth for puerperal septicæmia The diagnosis is established by the examination of the urine

**Course and Complications** The course depends largely on the treatment given Relapses are liable to occur or chronic pyelitis may supervene

**Prognosis** This is on the whole favourable

**Treatment** The patient should be put to bed and a saline purge given

*The Diet* The patient should take plenty of fluid (6 to 8 pints daily) such as water fruit juices and barley water Hot applications such as cataplasma kaolini should be placed on the loin if it is painful The urine should be made alkaline by giving a mixture of Sod bicarb gr 30 sod cit gr 30 pot acetat gr 30 syr aurant m 30 aquam ad fl oz 1 Fl oz 1 four hourly As soon as the urine is alkaline the

temperature usually falls and the mixture can be given six ho then 3 times a day as required The majority of infections resp to sulphonamides and antibiotics Sulphacetamide, chloram or aureomycin should be used for Bact coli infections The d sulphacetamide is four 0 5 G tablets followed by four tablets later and then two tablets t i d for 7 to 14 days The flui should be about 4 to 5 pints in the 24 hours and the potassium mixture given simultaneously The dose of chloramphenic aureomycin for an adult is two 0 5 G tablets eight hourly fo days Penicillin is of value in streptococcus fæcalis and staphy infections and streptomycin in pyocyaneus infections resistant case the sensitivity of the organism against the antibiotics should be tried Thus the Bact coli may be sens terramycin and insensitive to other antibiotics or the B proteu coli or Ps pyocaneus may be sensitive to Furadantin The dos adult weighing over 100 lbs is mg 100 t i d and if weighing o lb mg 100 q i d For a child the dose is mg 50 b i d or t i tablets are mg 50 or mg 100 Mandelic acid is of value in son of lower urinary tract infection It may be given as Mandelamin or four 0 25 G tabs t d s for 2 weeks A potassium citrate mixtu not be given with Mandelamine as the *p*H of the urine must b between 5 5 and 6 5

### Chronic Pyelitis

*(Relapsing or Quiescent Pyelitis)*

**Clinical Findings** Often there are no local symptoms b patient may complain of frequency of micturition and irregular a of fever with malaise

**Differential Diagnosis** This is established by ureteric cat sation An X ray examination should be made to exclude a calculus, and the urine also examined for tubercle bacilli

*Treatment* *It may be necessary to remove a calculus or reli* obstruction by operation before a cure can be obtained A cou sulphacetamide or of an antibiotic drug to which the organism is sen should be given as described above

### *Cystitis*

**Definition** Inflammation of the bladder

**Etiology** Cystitis is due to infection with organisms such a Bact coli the staphylococcus the streptococcus, the Neisseria g rhœa (gonococcus) the Mycobacterium tuberculosis (B tubercu and the Bacterium typhosum (B typhosus) It may be associated vesical bilharziasis a calculus adhesions between the bladder and or a diverticulum pyelitis enlargement of the prostate an ure stricture pelvic tumour retroverted gravid uterus spinal cord le etc *Predisposing causes* Chill, constipation and over indulgen alcohol and incomplete emptying of the bladder at micturition

Clinical Findings *Acute Cystitis* The patient is suddenly t

urine every 3 or 4 minutes, only about a teaspoonful being evacuated at a time There is usually no fever and no constitutional disturbance *Chronic Cystitis* The symptoms are less marked but there is often an aching pain over the pubes or in the perineum and frequency of micturition The urine An acid reaction implies infection with the Bact coli B tuberculosis or the gonococcus The urine is alkaline with the other infections and in any mixed infection It is cloudy contains a trace of albumin and blood may be present The deposit shows vesical epithelial cells and often some pus cells In children a staphylococcal infection is not uncommon The parent notices that the urine is thick or contains slimy or stringy matter which may only be present in the morning specimen

Treatment *Acute Cystitis* The patient should be kept in bed and hot applications placed over the hypogastrium The diet and regulation of the bowels are as for the treatment of pyelitis If the urine is acid an alkaline mixture should be given with tnc hyoscyami m 30 six hourly If it is alkaline acid sod phosph should be admini stered such as Acid sod phosph gr 30 to 60 tnc hyoscyam m 30 sp chlorof m 7 aquam ad fl oz 1 Fl oz 1 in water six hourly In many cases sulphacetamide in doses of 1 G t d s for 5 to 7 days or an antibiotic selected according to the sensitivity of the organism is very successful or Urolucosil 0 1 G tab one or two tablets 5 times daily If Urolucosil is given the fluid intake should be restricted for 5 days to 40 oz daily and no alkalis must be taken Surgical treatment may be required later to remove a causative condition

*Chronic Cystitis* In addition to the above measures the bladder may be irrigated with a 1/12 000 solution of silver nitrate and an autogenous vaccine given weekly beginning with small doses such as 250 000 organisms

## Carcinoma of the Prostate

Owing to the success attendant on the use of synthetic œ trogens the treatment of carcinoma of the prostate is passing into the hands of the physicians Stilbœstrol hexœstrol or dienœstrol may be pre scribed It is customary to begin treatment with stilbœstrol in doses varying from 1 to 15 mg a day the larger amounts being given in divided doses three times daily The effect is judged by the patient s general condition and symptoms and by the serum acid phosphatase content In the untreated case the acid phosphatase is raised to between 10 and 30 King Armstrong units or more the normal figure being less than 2 5 units per 100 ml

After a time perhaps 2 to 4 years the stilbœstrol may appear to lose its effect or nausea and vomiting may be troublesome symptoms The stilbœstrol may then be replaced by hexœstrol or dienœstrol in similar doses The patient usually gains weight loses his bone pains and prostatic symptoms and there is enlargement of the breasts the nipples becoming very tender with an overdose In many cases unfortunately after relief of symptoms for a few years the disease shows itself again and now fails to respond to treatment

### Pyelonephritis

**Definition** Inflammation and suppuration involving the renal pelvis and kidney substance

**Etiology** The infections may be ascending and secondary to pyelitis or blood borne in pyæmia, or due to lymphatic spread from an appendix or retroperitoneal abscess

**Pathology** Multiple abscesses may be seen on the surface and in the substance of the kidney, with inflammation of the pelvis

**Clinical Findings** The patient has usually the symptoms of pyelitis with periodical attacks of fever and rigors Chronic pyelonephritis is a cause of high blood pressure, especially in young women In some cases there is excessive loss of sodium chloride and water in the urine (salt losing nephritis) with a low blood pressure and the patient may be thought to be suffering from Addison's disease Acute cases occur which may prove fatal in a week or so The urine contains organisms albumin pus and blood

*Treatment* This is as for pyelitis Surgical drainage of the kidney is required in some cases Chronic pyelonephritis in children is more difficult to treat and may require a long course of sulphadimidine for the first 2 weeks the dose is 0 1 G /lb body weight a day, followed by a half dose for several weeks

### Pyonephrosis

**Definition** Distension of the renal pelvis with pus

**Etiology** Pyonephrosis results from infection of the renal pelvis together with obstruction to the outflow of urine The latter may be due to a stricture, enlarged prostate, diverticulum of the bladder vesical calculi, growth etc

*Pathology* One or both kidneys may be affected depending largely on the site of the obstruction

**Clinical Findings** The patient complains of malaise and usually of aching or pain in the loins

*On Examination* A renal tumour may be felt on one or both sides The temperature is usually raised The urine contains pus albumin and organisms If both kidneys are affected there is likely to be an increase in the blood urea and non protein nitrogen

**Treatment** This is surgical

### Perinephric Abscess

**Definition** Suppuration in the tissues around the kidney usually in the pararenal fat

**Etiology** The infection may be blood borne the focus being in a cutaneous boil or carbuncle or in the tonsils Lymphatic spread may occur from an infected kidney gall bladder appendix or from spinal caries

**Clinical Findings** The patient may complain of no symptoms except malaise due to fever and at times joint pains In other cases an aching or a sharp pain may occur in the loin

*On Examination* The temperature is found to be raised and it runs

an irregular intermittent course Tenderness is elicited in the loin and later a definite bulging may be seen or a swelling is felt on abdominal palpation over the kidney The patient may lie with the hip joint flexed on the affected side if the abscess is tracking downwards If on the other hand it is situated above the kidney the diaphragm on the affected side may be raised This results in some dulness and weak air entry over the base of the corresponding lung but tactile fremitus is present An X ray examination will also reveal the position of the diaphragm The blood A leucocytosis of about 12 000 to 20 000 per c mm may be present The urine contains a trace of albumin some pus cells and organisms (usually staphylococci) if the kidney is also affected The abscess may eventually point in Petit s triangle or rupture intraperitoneally

**Differential Diagnosis** The history of a crop of boils or carbuncles is suggestive but if there are no localising symptoms other causes of obscure pyrexia have to be considered and the diagnosis is not usually made until a swelling appears In the early stages the persistent fever joint pains and constipation are suggestive of infective endocarditis or of a Brucella abortus infection The agglutination test on the blood will exclude the latter

**Treatment** This is surgical the abscess being drained

## Genito urinary Tuberculosis

*Tuberculosis may affect the kidneys ureters bladder, or the genital* organs such as the epididymis testes seminal vesicles prostate Fallopian tubes and rarely the uterus and ovaries

### Tuberculosis of the Kidney

**Pathology** In the majority of cases the infection is blood borne ascending infection from the bladder being rare The primary focus may be in the lungs lymph glands or elsewhere but the site is often unrecognisable clinically

**Clinical Findings** The patient may complain of aching in the loin frequency of micturition and blood in the urine

**Differential Diagnosis** If the urine contains tubercle bacilli and pus cells renal tuberculosis is almost certainly present Cystoscopy and catheterisation of the ureters are necessary to establish the site of the lesion The presence of tubercle bacilli alone in the urine does not necessarily mean that progressive renal tuberculosis will inevitably ensue

**Treatment** Very successful results have been obtained by triple therapy streptomycin 1 G intramuscularly twice a week isoniazid mg 100 t i d by mouth and sodium para aminosalicylic acid (P A S ) 5 G t i d by mouth This should be continued for a minimum period of 1 year The patient should be in bed for 6 to 12 months Surgical treatment is not usually required for urinary tuberculosis but may be necessary in genital tuberculosis after a prolonged course of chemo therapy

### Tuberculous Cystitis

This is usually secondary to tuberculosis of the kidney epididymis or prostate In vesical tuberculosis treatment on the above lines often has an extremely good result in increasing the capacity of the bladder and so in reducing the frequency of micturition

### *Hydronephrosis*

**Definition** Dilatation of the renal calyces and pelvis

**Etiology** Hydronephrosis may be congenital or acquired, and is due usually to a gradual or intermittent obstruction to the outflow of urine

*Congenital Hydronephrosis* The obstruction may be caused by an abnormal branch of the renal artery, by a disordered function of the pelvi ureteral junction, or by an imperforate urethra It is usually bilateral

*Acquired Hydronephrosis* The causes may be grouped as follows 1 Renal A calculus or growth in the pelvis 2 Ureteric A disorder of the nervous control of the pelvi ureteral junction Kinking of the pelvi ureteral junction by a movable kidney or renal tumour A calculus or stricture Pressure due to a pelvic or abdominal tumour, or adhesions Injury during an operation 3 Vesical A calculus or tumour 4 Urethral A stricture calculus enlarged prostate or phimosis

**Pathology** The kidney substance atrophies and only a thin covering may remain In internal hydronephrosis the calyces are dilated but later the pelvis distends and the kidney substance may be replaced by a sac as large as a cocoa nut, the ureter also dilating to the level of the obstruction In intermittent hydronephrosis which is usually associated with a kinking of the ureter the pelvis dilates and contracts as the obstruction appears and disappears Bilateral hydronephrosis is due to obstruction in the urethra or bladder

**Clinical Findings** *Intermittent Hydronephrosis* The patient is usually a woman who complains of periodical attacks of pain in the *loin generally on the right side* and nausea and vomiting often associated with fever There is scanty micturition and relief comes with the passage of large quantities of urine The pain may be referred to the *healthy side by a reno renal reflex* Kinking of the ureter may give *rise to pain resembling renal colic which is known as a Dietl s crisis*

*On Examination* A renal tumour may be felt which disappears *with the onset of the polyuria*

*Continuous Hydronephrosis* The symptoms are less marked and there is aching in the loin If however the condition is bilateral and *progressive symptoms of chronic uræmia gradually ensue*

*On Examination* A renal tumour may be felt The blood A rise in the nitrogen figures indicates that both kidneys are affected

*Differential Diagnosis* The hydronephrosis may be demonstrated by pyelography If more than 20 ml of 15% sodium iodide can be injected into the renal pelvis hydronephrosis is held to be present *Radiography after intravenous injection of* Uroselectan B may also demonstrate the lesion

**Course and Complications** The course must vary with the nature of the obstruction Pyonephrosis may occur as a complication

**Prognosis** This also varies with the cause Bilateral lesions prove fatal unless the obstruction is removed before irreparable and extensive renal damage has been done death occurring from uræmia or suppuration

**Treatment** If the hydronephrosis is due to a movable kidney a renal support should be tried If this does not afford relief an operation will probably be necessary In the majority of cases the treatment is surgical

## Nephrolithiasis

## (*Renal Calculus*)

**Etiology** The cause of calculus formation is not known Certain factors undoubtedly play an important part These include infection urinary stasis a concentrated urine and the presence of irreversible colloids such as fibrin which after precipitation will not go back into solution Overdosage with alkalis as in the treatment of peptic ulcer may lead to the formation of phosphatic calculi It is not known whether calculi are associated with a deficiency of vegetables in the diet and it is uncertain why they are prone to occur in England in certain localities as in Norfolk In India stone occurs amongst the poorer members of the community who live chiefly on cereals and do not have sufficient milk This is possibly due to lack of vitamin A They are not believed to be associated with chalky water Owing to decalcification of bones calcium phosphate calculi may form in hyper parathyroidism or more rarely in hyperthyroidism They may occur without any bony lesion Lying in bed for a long time in the treatment for example of a fractured femur or poliomyelitis or trauma to the kidney is responsible for calculus formation in some cases Sulphon amides may also result in renal calculus formation if sufficient fluid is not taken during their administration The acetylated form of the sulphonamide is precipitated in the kidney Continued over-dosage with vitamin D may also lead to the formation of renal calculi Calculus formation usually occurs after the age of 20 males being most often affected but urates may be deposited in the kidneys of infants and vesical calculi found in the bladders of poorly nourished children Occasionally a large calculus is found in the kidney of a child under the age of 2 years One composed of sodium magnesium and ammonium phosphate and another of xanthine have been removed

**Pathology** Calculi usually form around a colloid nucleus such as fibrin mucopus cell *débris* and micro organisms Primary calculi are said to form in acid urine without any bacterial inflammation whereas secondary calculi are deposited in alkaline urine infected with organisms Thus a phosphatic crust may be formed around an uric acid nucleus The following varieties are described *Uric acid* Brown hard and irregular *Calcium oxalate* (mulberry calculus) Dark and irregular When formed in the bladder they are round they may be dendritic when occurring in the renal pelvis They are often mixed with calcium phosphate or uric acid *Ammonium urate* Hard and brownish *Triple*

*hosphate* Rather soft and smooth *Mixed or laminated calculi* They ave an uric acid nucleus and a phosphatic coating *Cystine* Soft ellow green, radially laminated *Xanthine* Reddish brown *Indigo* lue, will mark paper It is derived from indol and is extremely rare *alcium carbonate* Smooth hard and dark grey *Urostealith* Con uning cholesterin

**Clinical Findings** If the calculus remains in the kidney substance iere may be no symptoms or the patient complains of aching in the in, and albumin and blood are found in the urine These symptoms iay be related to jolting If the calculus obstructs the outflow of urine iere is severe pain owing to stretching of the renal capsule This may cur with a stone in the ureter and the pain is due to this distension ither than to the passage of the stone along the ureter The pain ases either when the kidney does not secrete more urine or when the ostruction is relieved The pain is felt in the flank and may be referred the sound side it may also radiate to the groin and inner side of the igh There is usually a sense of nausea and vomiting may occur enal colic is an agonising pain, which is probably caused by muscular oasm of the renal pelvis and ureter The pain occurs in paroxysms id radiates along the course of the ureter to the groin hypogastrium id testicle and the testicle may be drawn up The patient is pale or ushed sweats rolls about in agony and may vomit There is a equent desire to micturate and the urine is scanty and may contain ood The attack lasts usually from a few minutes to 2 hours but it iay be considerably longer After the attack there is aching and nderness in the loin

*On Examination* During the attack there is abdominal rigidity on ie affected side The chief symptoms of a vesical calculus are attacks pain felt at the end of the penis after micturition radiating to the rineum and inner side of the thigh There is also frequency of icturition and the urine shows evidence of cystitis

*Differential Diagnosis* Renal colic must be differentiated from her varieties of abdominal colic and pain such as biliary intestinal pendicular or pancreatic colic or from a Dietl's crisis associated with movable kidney and kinking of the ureter from a twisted ovarian cyst rarely from a tabetic crisis The X rays will show renal calculi ovided they contain a sufficiency of calcium (see Fig 43) Other adows such as those caused by calcified glands and phleboliths must excluded Pyelography may thus be required in order to localise e shadow accurately

**Course and Complications** Several attacks of renal colic are not frequent The chief complications are due to 1 *Infection* resulting pyelitis pyelonephritis pyonephrosis and cystitis 2 *Obstruction* oducing hydronephrosis pyonephrosis renal atrophy and anuria if th kidneys are put out of action 3 *Ulceration* with extravasation urine through the ureter A stricture may subsequently form *Malignant disease* affecting the kidney

**Prognosis** In some cases only one attack of renal colic occurs but e prognosis is always serious as the calculi tend to recur even after



[illegible]

*phosphate* Rather soft and smooth *Mixed or laminated calculi* They have an uric acid nucleus and a phosphatic coating *Cystine* Soft yellow green, radially laminated *Xanthine* Reddish brown *Indigo* Blue will mark paper It is derived from indol and is extremely rare *Calcium carbonate* Smooth hard and dark grey *Urostealith* Containing cholesterin

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*On Examination* During the attack there is abdominal rigidity on the affected side The chief symptoms of a vesical calculus are attacks of pain felt at the end of the penis after micturition radiating to the perineum and inner side of the thigh There is also frequency of micturition and the urine shows evidence of cystitis

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**Course and Complications** Several attacks of renal colic are not infrequent The chief complications are due to 1 *Infection* resulting in pyelitis pyelonephritis pyonephrosis and cystitis 2 *Obstruction* producing hydronephrosis pyonephrosis renal atrophy and anuria if both kidneys are put out of action 3 *Ulceration* with extravasation of urine through the ureter A stricture may subsequently form 4 *Malignant disease* affecting the kidney

**Prognosis** In some cases only one attack of renal colic occurs but the prognosis is always serious as the calculi tend to recur even after

operative removal The stone may be passed without an operation If both kidneys are affected or if there is evidence of failure of renal function the outlook is very grave

Treatment *Prophylactic* Plenty of fluids should be drunk in hot climates Pyelitis should be adequately treated

*Curative During the attack* Local heat applied to the loin or a hot bath may relieve the pain of a mild attack For a severe attack an injection should be given of morphin sulph gr 1/3 and atropin sulph gr 1/100 If the pain is not relieved inhalation of chloroform may be required *After the attack* To aid the passage of the calculus along the ureter a mixture containing tnc belladon m 15 and pot citras gr 80 should be given every 6 hours until the pupils dilate If the stone is not passed the opinion of a surgeon should be obtained as to the advisability of operative removal In any case the patient should be kept in bed until the hæmaturia has ceased and an alkaline mixture given if the urine is acid containing Pot cit gr 30 pot bicarb gr 20 sod bicarb gr 20 sp chlorof m 7 aquam ad fl oz 1 Fl oz 1 t d s *Subsequent treatment* If oxalate or urate crystals are present in the urine a dose of the alkaline mixture should be given at night sufficient to render the morning specimen of urine alkaline The diet should be rich in vegetables and if urates are passed meat should only be taken in small quantities and substances rich in nucleo protein such as sweetbreads kidneys and liver should be avoided The diet recommended for oxaluria is described on p 463 In all cases at least 4 to 5 pints of bland fluids should be taken in the 24 hours

## Movable Kidney

*(Floating Kidney Nephroptosis)*

Undue mobility of the kidney is not of great clinical importance Three degrees are described palpable movable and floating The patient is usually a middle aged woman and the right kidney is generally affected The patient may complain of aching in the loin or of attacks of severe pain in the loin radiating along the course of the ureter to the groin or inner side of the thigh (Dietl s crisis) Micturition is scanty and there may be hæmaturia This is probably due to kinking of the ureter An intermittent hydronephrosis may develop In some cases relief is obtained by wearing a belt with a kidney pad but operation may be necessary in cases of Dietl s crises

## Congestion of the Kidneys

Passive congestion occurs in heart failure (cardiac kidney) or is due to obstruction of the renal veins The urine is diminished in volume the specific gravity is raised albumin is present and red cells granular and hyaline casts may be found in the deposit

## Infarction of the Kidneys

The emboli are usually derived from the heart Minute bacterial emboli in subacute infective endocarditis give rise to focal nephritis Larger infarcts cause severe pain in the kidney region with hæmaturia

If blood is present in the urine catheterisation of the ureters will show from which kidney it is coming

**Prognosis** Death usually occurs from 1 to 2 years from the date of diagnosis

**Treatment** The results of nephrectomy or of X ray treatment are disappointing

## Renal Cysts

The following varieties of cysts may occur in the kidneys Congenital cystic kidney Solitary cysts Retention cysts Hydatid cysts Degeneration cysts in new growths

### Congenital Cystic Disease of the Kidneys

### (*Polycystic Disease*)

**Etiology** There are two types of the disease infantile and adult In both the cysts are glomerular and appear as closed systems In the infantile type there is no morphological evidence of a connection between the cysts and the renal pelvis In the adult type cysts appearing along the course of the tubules as a rule are connected with the excreting tubules and renal pelvis The etiology is obscure In the infantile type the theory that there is a failure of the tubule of the nephron to unite with the collecting tubule may hold good but this is not so in the adult type The cystic nephron of the adult retains part of its functional activity and takes part in the formation of urine This would explain why the adult with the disease may survive until late middle life

**Pathology** The kidney may be considerably enlarged so much so that when occurring in the fœtus it causes difficulty in labour The kidney substance may be almost entirely replaced by cysts They contain a clear or turbid fluid in which urea and blood may be present One or both kidneys may be affected In addition cysts may be found in the liver and pancreas and at times in the lungs

**Clinical Findings** The infant may be still born or may die rapidly from uræmia If occurring in adults no symptoms are usually noted until after the age of 30 The symptoms are then those of malignant renal sclerosis or of a renal tumour The patient complains of lassitude headaches attacks of nausea and vomiting or of an aching in the loin

*On Examination* An irregular renal tumour may be felt on one or both sides The heart is usually enlarged the arteries thickened and blood pressure raised The urine resembles that of nephrosclerosis the volume being increased the specific gravity low and a trace of albumin being found with an occasional hyaline or granular cast Periodical attacks of hæmaturia may occur As the lesion progresses the blood shows retention of urea and non protein nitrogen

**Differential Diagnosis** The diagnosis cannot usually be established unless the irregular knobbly kidney is palpable Retrograde pyelo graphy shows characteristically lengthening and compression deformity of the pelvis and calyces of the dragon' or spider type The calyces and pelvis may show clear cut crescentic indentations some

### Syphilis of the Kidneys

Chronic nephrosis may occur in the secondary stage Later gummata may form with an associated amyloid degeneration of the kidneys

### Renal Tumours

*Simple Tumours* These are comparatively rare They include a *fibroma adenoma lipoma* papilloma and angioma An angioma of the renal pelvis may give rise to severe and so called " essential " hæmaturia A papilloma of the pelvis tends to be locally malignant recurring after removal and spreading down the ureter

*Malignant Tumours* These may be primary, and include a carcinoma such as an adenocarcinoma (hypernephroma or Grawitz *tumour) A squamous epithelioma may form in the renal pelvis The* primary sarcoma includes an embryoma (Wilm s tumour, containing muscle fibres and rarely cartilage and bone) Secondary carcinoma and secondary melanotic sarcoma also occur

**Pathology** The hypernephroma This is now usually considered to be a papillary adenocarcinoma arising in the renal tubules It *may occur in any part of the kidney It shows yellowish fatty areas* and hæmorrhages or cysts may be present Secondary deposits form in the long bones such as the tibia in the bodies of the vertebræ and in other organs, such as the lungs liver and brain The bony deposits are often very vascular Other varieties of carcinoma include an alveolar adenocarcinoma and a tubular adenocarcinoma

**Clinical Findings** *The patient may be an infant, in which case the* tumour is usually a sarcoma or hypernephroma

*On Examination* The child is weak pale wasted and the abdomen is swollen A renal tumour may be present The characteristic signs of this are A tumour is felt in the loin which enlarges downwards and backwards and later extends towards the mid line It is dull on per*cussion the dulness extending backwards to the flank* A band of colonic resonance may be found running across it The tumour has a rounded border and the upper pole may be palpable There is very slight movement with respiration The urine may contain blood or albumin

In an adult a hypernephroma may be so small that it gives rise to no local signs or it may form a definite swelling Pyelograms may reveal that one kidney is abnormal An aortogram may reveal the presence of a renal tumour with masses of tortuous arteries Painless hæmaturia is the earliest sign of a renal tumour in the majority of cases In the male the presence of a varicocele occurring for the first time in a patient of middle age which does not disappear on lying down is very suggestive of a malignant renal tumour especially if it is right sided Fever may occur apart from evidence of infection with an increased sedimentation rate of the red cells Secondary deposits are likely to form in the bones especially in the skull vertebræ femur and humerus Secondary deposits in the lungs usually give rise to no symptoms but X ray examination reveals the presence of soft woolly shadows in both lung fields

## CHAPTER VI

# FLUID AND ELECTROLYTE BALANCE

Disturbances of water and electrolyte metabolism may produce serious and irreversible illness It is important therefore to understand the basic principles of fluid and electrolyte physiology so that these disorders may be recognised corrected and if possible prevented

### Basic Physiological Considerations

The total body water of a man weighing 11 stones is approximately 49 litres or 70% of the body weight This is made up as follows —

| | | | |
|---|---|---|---|
| Intracellular fluid | 35 litres | | |
| Extracellular fluid | 14 litres | Tissue fluid | 10 5 litres |
| | | Blood plasma | 3 5 litres |

**Normal Daily Fluid Balance** The normal healthy adult on a normal diet in a temperate climate has the following water balance —

| *Intake (ml )* | | *Output (ml )* | |
|---|---|---|---|
| Fluid by mouth | 1 200 | Urine | 1 200 |
| Water content of food | 1 000 | Invisible loss (lungs and skin) | 1 200 |
| Water of oxidation | 300 | Stools | 100 |
| | 2 500 | | 2 500 |

Thus the fluid intake in such a person should equal approximately the urine output This does not usually apply in other circumstances for instance in febrile patients and in hot climates the invisible loss may be much greater

**Normal Electrolyte Balance** The blood plasma normally has an equal amount of acid and base each adding up to a total of 155 milli equivalents per litre (mEq /L ) The milli equivalent per litre is obtained by dividing the figure in mg per litre by the atomic weight of the ion concerned and multiplying this figure by the valency Below is given a list of the common ions together with their conversion figures —

| | | | |
|---|---|---|---|
| Na | mg per 100 ml | × 10 divided by | 23 |
| K | , | × | 39 |
| Ca | ( | × | 40) × 2 (valency) |
| Mg | ( | × | 24) × 2 |
| Cl | | × | 35 |
| $HPO_4$ | ( | × | 31) × 1 8 |
| $SO_4$ | ( | × | 32) × 2 |

Alkali reserve $CO_2$ vols per 100 ml divided by 2 22
Plasma protein G per 100 ml × 2 43

times with a half shadow due to projection of the cyst into the calyx or pelvis These changes are bilateral An operation may be performed *for a renal tumour*, and the true nature of the lesion may only be discovered in this way

**Course and Complications** The course is progressive, with gradual *failure of renal function* Polycystic degeneration of the liver and pancreas follows in some cases Cerebral hæmorrhage may occur as a complication which may be due to rupture of an aneurysm of the circle of Willis

**Prognosis** Death may ensue in from 3 to 25 years after congenital cystic disease has been diagnosed The patient may live for many years with a high blood nitrogen content

**Treatment** This is as for arteriolar nephrosclerosis The kidney should not be removed by operation

## The Solitary Cyst

This is usually met with in children It is probably a variety of polycystic disease The cyst is generally situated in the renal cortex and may contain over 2 pints of fluid In adults large solitary serous cysts occur in the parenchyma of the kidney They contain clear yellow fluid

## Retention Cysts

These are met with post mortem in cases of chronic nephritis *Clinically they give rise to no symptoms*

## Hydatid Cyst

*This is due to an echinococcus infection* If the cyst ruptures into the renal pelvis there is renal colic and hooklets are found in the urine It may also burst into the intestine or peritoneal cavity

## Cystic Degeneration of a New Growth

This is only discovered at the autopsy

## Fusion of the Kidneys

This usually occurs at the lower poles constituting the horse shoe kidney Less often fusion of the upper poles is present or the upper pole of one kidney unites with the lower pole of the other or both kidneys fuse to form the cake kidney

in order to excrete a given amount of waste products This in turn alters electrolyte excretion in the urine

### Types of Disturbances

These may take the form of excessive intake deficient intake excessive loss diminished loss or a mixture *e g* in pyloric stenosis there is both excessive loss and diminished intake

Three of the common types of upset of electrolytic balance are illustrated in the diagram (see Fig 45) Gastric juice usually contains

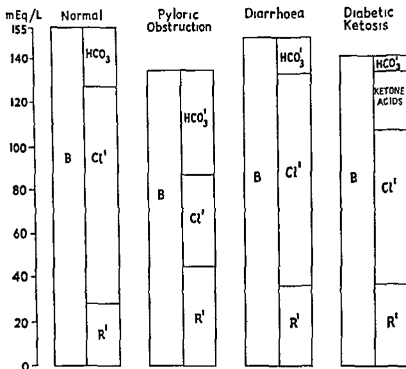



FIG 45 ACID BASE COMPOSITION OF BLOOD PLASMA IN VARIOUS CONDITIONS B represents total base R represents sulphate and phosphate radicals organic acids and plasma proteins (After Gamble James L)

free HCl and hence in any condition where there is vomiting more Cl will be lost than Na In pyloric obstruction therefore in addition to loss of base due to dehydration and increase in R (due to increased concentration of plasma proteins because of reduction of plasma volume resulting from dehydration and retention of phosphates and sulphates) the total mEq /L will be less than 155 More Cl is lost than base Na and this is compensated for by an increase in $HCO_3$ compartment The end result therefore is water deficiency diminution of base hypo chloræmia and alkalosis

In diarrhœa there is loss of water and also of Na K and Cl As pancreatic secretions contain excessive amounts of Na more Na is lost

Thus to convert a normal serum calcium level of 10 mg per 100 ml to mEq /L $\frac{10 \times 10}{40} \times 2 = 5$

The acid base composition of normal plasma is as follows (see also Fig 44) —

| *Base mEq /L* | | *Acid mEq /L* | |
|---|---|---|---|
| Na | 142 | $HCO_3$ | 27 |
| K | 5 | Cl | 103 |
| Ca | 5 | $HPO_4$ | 2 |
| Mg | 3 | $SO_4$ | 1 |
| | | Organic acids | 6 |
| | | Plasma protein | 16 |
| | 155 | | 155 |

The type of diagram (see Fig 44) introduced by Gamble forms a convenient basis for working out electrolytic problems The acid and base columns must always add up to the same total figure If this is less than 155 mEq /L the plasma is hypotonic as occurs when the loss of electrolytes is excessive and insufficiently replaced A total greater than 155 mEq /L is rare and occurs in such conditions as alkali alkalosis in the absence of much vomiting

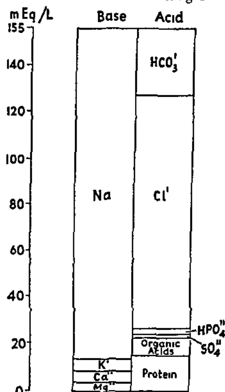



FIG 44 ACID BASE COMPOSITION OF BLOOD PLASMA (After Gamble James L Chemical Anatomy Physiology and Pathology of Extracellular Fluid sixth edition Harvard University Press Cambridge Massachusetts 1954 )

The daily requirement of Na as NaCl is 4 to 10 G (69 to 173 mEq ) most being excreted in the urine (an average figure being 110 mEq in 24 hours) and very little is lost in the fæces and in the sweat The daily requirement of K is 2 to 4 G (51 to 102 mEq ) and again 80% to 90% of it is excreted in the urine in cases of diarrhœa however much K may be lost in the stools Most of the sodium in the body is in the extracellular fluid whereas potassium is largely an intracellular ion

In healthy individuals the kidneys maintain an excellent control of fluid and electrolytes but conditions such as renal failure dehydration and shock may upset this considerably Impairment of concentrating power of the kidneys leads to larger amounts of obligatory urine than normal

remembered that urinary chloride may be present with low plasma levels in Addison's disease, pyloric stenosis with potassium deficiency, salt losing nephritis and conditions in which sodium loss has been greater than chloride loss, *e g* intestinal fistulæ. Chloride may be absent in the urine when salt depletion has been relieved and even when œdema is present, *e g* in hypoproteinæmia and depressed renal function with reduced glomerular filtration.

While most cases of electrolytic disturbance are mixed, it is convenient, if somewhat artificial, to consider the symptomatology of the commoner types of upset.

1 *Water Intoxication* Throbbing headache, hypertension, confusion, hallucinations and convulsions.

2 *Water Deficiency* Thirst, dehydration, scanty urine with a high specific gravity, urinary chlorides often increased, blood pressure normal until late, plasma chlorides normal or raised and blood urea only slightly raised.

3 *Salt Depletion* Thirst is absent. Dehydration, lassitude, cramps, vomiting, normal amount of urine until late with a low specific gravity, urinary chlorides usually absent, hypotension and fainting with a considerably raised blood urea.

4 *Sodium Intoxication* Œdema, raised jugular venous pressure and left ventricular failure.

5 *Potassium Deficiency* Muscular weakness, paralysis, decreased tendon reflexes and electrocardiographic changes, namely a prolonged Q–T interval with flat or inverted T waves.

6 *Potassium Intoxication* Muscular weakness, ventricular fibrillation and auricular arrest. The electrocardiogram shows the changes described on p 481.

**Treatment** This is directed at correction of previous losses and present abnormal losses together with maintenance of basic daily requirements of fluid and electrolytes. Intravenous therapy is often necessary, particularly if the patient is vomiting. Basic requirements of fluid and salt in 24 hours can be given as 1 litre of normal saline and 1½ to 2 litres of 5% dextrose (which electrolytically amounts to giving water) or as 2½ to 3 litres of 1/5 normal saline with 4 3% dextrose.

In estimating the extra electrolytic requirements which need to be added to the above fluids, the following are rough guides:—

1 For every 100 mg (17 mEq /L) the plasma Cl needs raising to normal (585 mg /100 ml or 103 mEq /L) give 0 5 G (8 5 mEq) of NaCl per kg of body weight.

2 For every integer the specific gravity of the plasma is above 1 027 give 200 ml of fluid, usually as 100 ml of normal saline and 100 ml of 5% dextrose, assuming a mixed water and salt deficiency.

3 For each 1 mEq the plasma bicarbonate has to be raised to normal level (27 mEq /L) give 4 4 ml of 1/6 M sodium lactate per kg of body weight, provided the acidosis is a metabolic one and the *p*H of the blood is decreased.

Normal saline contains 9 G of NaCl per litre. Although it is isotonic with the blood, it is by no means physiological as each litre

than Cl and B is diminished more than Cl′ R is increased once more and so $HCO_3$ is diminished giving rise to acidosis A similar type of electrolyte and water disturbance occurs in intestinal obstruction

In diabetic ketosis both columns are reduced due to renal disability caused by rapid dehydration In the acid column ketone acids are present and this together with an increase in R occurs at the expense of the bicarbonate so that the patient is acidotic as well as hypo chloræmic

### Causes of Electrolytic Disturbances

1 *Salt Depletion* Pure salt depletion is commonly due to abnormal loss of water and electrolytes with subsequent replacement of water alone Salt depletion also occurs in renal disease such as chronic nephritis and pyelonephritis acute tubular necrosis and in renal dysfunction due to hormonal disturbances as in Addison s disease and hypopituitarism, as the kidney does not reabsorb sodium normally Excessive loss of salt and water in the urine occurs in diabetes mellitus with ketosis Extrarenal loss occurs in chronic diarrhœa intestinal fistulæ vomiting and sweating

2 *Water Depletion.* This is less common than salt depletion It occurs in patients who cannot or will not drink fluids *e g*, dysphagia old age or coma Mixed salt and water depletion is of course more frequent than either pure salt or pure water lack

3 *Sodium and Water Excess* This occurs in hypoproteinæmia the nephrotic syndrome congestive heart failure acute nephritis cirrhosis of the liver therapy with cortisone, ACTH DOCA and testosterone It also occurs in excessive progesterone secretion (premenstrual)

4 *Potassium Depletion* Conditions under which this is liable to occur include diarrhœa continued aspiration of gastro intestinal contents vomiting starvation (as the kidney is unable to conserve potassium as efficiently as it conserves sodium and there is breakdown of the protoplasmic tissues releasing potassium) diabetic ketosis in the recovery phase and Cushing s syndrome It may also occur during therapy with ammonium chloride the steroid hormones and para aminosalicylic acid The rare condition of primary aldosteronism results in excessive excretion of potassium in the urine

5 *Potassium Excess* This is found in patients with oliguria or anuria due to acute tubular necrosis chronic renal failure burns or circulatory failure It also occurs in Addisonian crisis

### Assessment of Type of Disturbance

In each case of suspected electrolytic disturbance it is essential to take a clear history and make a careful examination An accurate record of fluid intake and output is also necessary Estimation of plasma Na K Cl and alkali reserve together with a blood urea are also important The plasma specific gravity (normal 1 027) in the presence of normal plasma proteins is also a useful indication of the degree of dehydration Simple bedside estimation of the amount of urinary chloride is also most useful as a guide to the plasma level provided it is

## CHAPTER VII

# THE HÆMOPOIETIC AND RETICULO-ENDOTHELIAL SYSTEMS

Introductory The hæmopoietic system is concerned with the production during life of the formed elements of the blood According to the polyphyletic view the red cells and granulocytes are produced by the bone marrow the lymphocytes develop in lymph glands and lymphatic tissues generally and the monocytes are derived from the reticulo endothelial system The platelets are derived from mega karyocytes in the bone marrow Certain factors are required for the maturation of the red cells The primitive marrow cell (hæmocytoblast) is converted into a proerythroblast (megaloblast) possibly with the aid of an unknown agent The active principle from the liver and stomach and perhaps the vitamin B complex aid in the change from proerythroblast to normoblast Iron copper thyroxine and possibly vitamin C are concerned with the change from normoblast to erythrocyte The reticulo endo thelial cells are branched connective tissue cells with affinity for special dyes such as pyrrhol blue They are widely distributed in the spleen liver bone marrow omentum adrenals the pituitary etc Red cell destruction is effected by the reticulo endothelial cells in the spleen and liver and possibly elsewhere The total blood volume is approximately 5 litres

The following examinations of the blood or bone marrow may be required in the investigation and treatment of diseases in this group

1 *A Blood Count* A normal blood count for an adult is as follows Red cells average for both sexes 5 000 000 per c.mm Hb average normal for males and females Haldane standard 105% Sahli standard 85% These figures are equivalent to 14 5 G Hb per 100 ml blood The size shape and staining properties of the red cells are noted Normally no nucleated red cells are seen except in an infant for a few days after birth Reticulocytes are immature red cells showing with vital stains a reticulum in their cytoplasm and normally present to the extent of ½ to 1% They are the same cells as exhibit polychromasia when stained in a dry film An excess of reticulocytes in the blood indicates that the red cells are being rapidly put forth into the circula tion The hæmoglobin content of each average red cell is indicated by (a) *The Mean Corpuscular Hb* This is the weight of Hb in each average cell The normal mean is 29 5γγ (γγ = 0·000000001 mg) (b) *The Mean Corpuscular Hb Concentration* This indicates the degree to which each red cell is saturated with Hb The normal mean is 85% (c) *The Colour Index* This indicates the amount of Hb in each average red cell and taking as arbitrary normal figures Hb 100% and red

contains 154 mEq of Na and Cl which is higher than the normal plasma level Hence it cannot be given indiscriminately as if it were truly a physiological solution although in the presence of normal renal function the excess chloride is usually excreted in the urine

Potassium supplements should be given by mouth or intravenously in the absence of an adequate urinary output, *e g* , 1,500 ml per day The basic daily requirements are 60 mEq , and this may be given by mouth as 3 to 5 G of KCl in the absence of any food intake Intravenously it may be given as 2 23 G of KCl per litre in two of the 3 litres of fluid given each day It should never be given at a rate faster than 1 litre of such fluid in 3 hours Additional potassium must be administered if there are previous losses In this connection it is important to remember that in the absence of an adequate calorie intake the daily loss of potassium in the urine will be increased due to breakdown of protoplasmic tissue A useful potassium mixture for oral use consists of 1 G each of potassium acetate bicarbonate and *citrate made into a mixture and given 2 to 3 times a day to cover* basic requirements It is less unpleasant for the patient to take than is KCl

In conclusion it must be stressed once more that for correction of fluid and electrolyte losses it is essential to have an accurate record of fluid and electrolyte intake and output

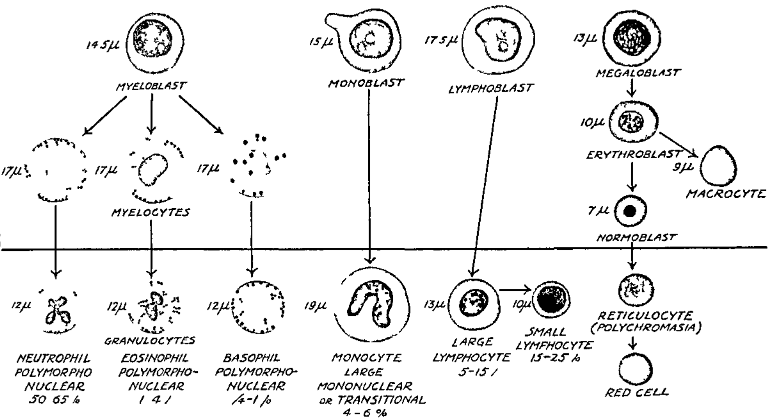



Fig 46 The Blood Cells and their Precursors ( × 1 000) The Cells below the Line are Normally Present in Blood

[T f ep 04

cells 5 millions, the C I is $\frac{\text{Hb }\%}{\text{Red Cells}\%}$ The normal figure is 1 (d) 
*Saturation Index* This indicates the degree to which each average cell is saturated with Hb, as shown by comparing the mean corpuscular Hb concentration of the blood in question with the normal mean corpuscular Hb The normal saturation index is 1

The size of each average red cell is indicated by (a) *The Mean Corpuscular Diameter* The normal average is 7 2μ (b) *The Mean Corpuscular Volume* The normal average is 87cμ (c) *The Volume Index* This indicates the volume of an average red cell of the blood in question compared with the normal average red cell volume The normal figure is 1 The average life of a red cell is about 120 days it is destroyed by disintegration White cells 6 000 to 9 000 per c mm Differential white count Granulocytes These include Polymorphonuclears (neutrophils) 50 to 65% eosinophils 1 to 4% mast cells (basophils) ½ to 1% Lymphocytes (small) 15 to 25% Lymphocytes (large) 5 to 15% Large mononuclears (monocytes hyaline or transitional cells) 4 to 6% (see Fig 46) The average life of a polymorphonuclear cell is four days and of a lymphocyte less than a day

2 *A Platelet Count* Normally there are 200 000 to 500 000 platelets per c mm

3 *Fragility of the Red Cells* Normally hæmolysis does not occur in solutions of sodium chloride, until the percentage of NaCl has been lowered from that of normal saline (0 9%) to a strength of 0 45% With increased fragility hæmolysis may begin at 0 65% NaCl and be complete at 0 5%

4 *The Bleeding Time* A small cut is made in the ear or finger Drops of blood are taken up on absorbent paper every 30 seconds until bleeding stops The normal time is 1 to 5 minutes

5 *The Coagulation Time* This varies with the method used, and a control should always be made to allow a normal reading as comparison The normal time by Lee and White's method is 4 to minutes

6 *The Sedimentation Rate of the Red Cells* By the Westergren method in which the tube is divided into 200 mm the normal reading at one hour is plasma 1 to 5 mm for men and for women plasma 4 to mm By Wintrobe s method the normal sedimentation reading at one hour is plasma 0 to 9 mm for men and for women plasma 0 to 15 mm

7 *The Estimation of Prothrombin in Plasma* The clotting time of oxalated plasma when mixed with an excess of thrombokinase and an optimum amount of calcium is employed as a direct measure of the prothrombin content of the plasma Using the method of Quick normal plasma (100% prothrombin) should clot in 12 to 13 seconds The concentration in percentage can be calculated from a concentration curve, thus 30% = approximately 24 seconds

8 *Sternal Puncture* 0 25 c c of marrow fluid is removed by puncture of the sternum using a Salah needle and dry syringe The fluid is put in a tube containing Wintrobe s dry oxalate mixture, smears are then stained by Leishman's method

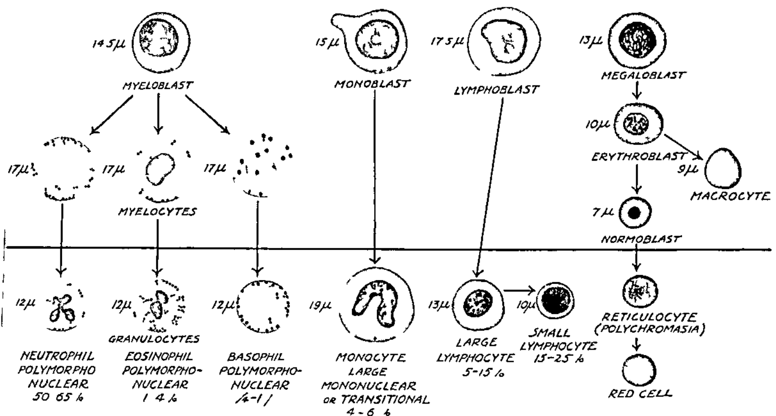



Fig 40 The Blood Cells and their Precursors ( × 1 000) The Cells below the Line are normally present in Blood

[To face p 04]



**Cells in Normal Bone Marrow Smears**

*(Percentages)*

| | | | |
|---|---|---|---|
| Hæmocytoblasts | | | 0 to 1 |
| Proerythroblasts | | | 0 to 4 |
| Early and intermediate normoblasts | | | 4 to 15 |
| Late normoblasts | | | 7 to 19 |
| Myeloblasts | | | 0 to 2 5 |
| Promyelocytes | | | 0 5 to 5 |
| | *Neutrophil* | *Eosinophil* | *Basophil* |
| Myelocytes | 2 to 8 | 0 to 1 | |
| Metamyelocytes | 10 to 25 | 0 to 2 5 | |
| Polymorphs | 10 to 40 | 0 to 4 | 0 to 1 |
| Lymphocytes | | | 5 to 20 |
| Monocytes | | | 0 to 5 |
| Plasma cells | | | 0 to 1 |

Total cell count 25 000 to 100 000 per c mm
Myeloid to nucleated red cells ratio 2 1 to 8 1

9 *Blood Grouping* The red cells may contain agglutinogens A or B or both or none and the serum may contain agglutinins $\alpha$ or $\beta$ or both or none Agglutinin $\alpha$ or $\beta$ causes agglutination of corpuscles containing agglutinogen A or B respectively Four Landsteiner groups are described according to the corpuscular agglutinogen content

| *Group* | *Corpuscular Agglutinogen* | *Serum Agglutinin* |
|---|---|---|
| A B or 1 | A B | 0 |
| A or 2 | A | $\beta$ |
| B or 3 | B | $\alpha$ |
| 0 or 4 | O | $\alpha + \beta$ |

There are also Landsteiner sub groups such as $A_1$ $A_2$ $A_1B$ and A B Further the serum from group B donors may contain $\alpha$ $\alpha_1$ and $\alpha$ agglutinins The Rhesus factor is described on p 96 Blood grouping has become more complicated by the discovery of other factors such as the Lutheran Kell M N P and S factors which may cause incompati bility

*Donors* Group AB may give blood to Group AB Group A to A or AB Group B to B or AB and Group O is a universal donor *Recipients* Group AB is a universal recipient Group A may receive blood from A or O Group B from B or O and Group O from O It is better to use the blood of a donor of the same group as the recipient rather than that of a universal donor Further the serum of the recipient should always be tested against the corpuscles of the donor

*The Coombs Test* This is used to demonstrate the presence of antibodies adsorbed on to red cells or bacteria The direct test is of value in the diagnosis of erythroblastosis fœtalis and acquired hæmolytic anæmia and in demonstrating cold agglutinins The indirect test shows the presence of Rh antibodies in low titre It is used in Rh

typing and in determining the Duffy blood group In Brucellosis by doing the indirect test using the patient's serum and bacteria immune bodies are adsorbed on the bacteria

Various abnormal cells may appear in the blood in disease 1 *Abnormal red cells* Macrocytes are large non nucleated cells Microcytes are small non nucleated cells Megaloblasts and erythro blasts are large nucleated cells Normoblasts are normal sized nucleated cells The red cells may exhibit polychromasia (staining diffusely a bluish colour) or punctate basophilia (showing blue granules) Target cells have a darkly stained central area Red cells may be irregular in size (anisocytosis) or in shape (poikilocytosis and sickle cells) 2 *Abnormal white cells* Precursors of granulocytes These are premyelocytes with oval nuclei, myelocytes with indented nuclei and metamyelocytes with a lobed nucleus These cells may contain neutrophil eosinophil or basophil granules and are classed accordingly The parent cell of the myelocyte is called a myeloblast and that of the lymphocyte is a lymphoblast (see Fig 46) It is often very difficult to distinguish between a large immature lymphocyte (lymphoblast) and a myeloblast The myeloblast is slightly smaller and often the oxidase test shows that the cytoplasm is granular In the very early stages however the oxidase test is negative The oxidase test is negative with the lympho blast the cytoplasm being agranular

## Leucocytosis

**Definition** An increase in the number of white cells in the blood

**Etiology** Physiological leucocytosis occurs after meals exercise cold baths and during labour and the first week of the puerperium In children there is a physiological lymphatic leucocytosis In patho logical leucocytosis the different types of white cell may be unequally affected *Polymorphonuclear leucocytosis* This is met with in infections in lobar pneumonia in paroxysmal tachycardia in coronary occlusion in diabetic coma and after an acute hæmorrhage 90% of the white cells may be neutrophil polymorphonuclears *Lymphatic leucocytosis* This occurs in lymphatic leukæmia glandular fever whooping cough and sometimes in mumps Up to 90% of the white cells may be lymphocytes *Eosinophilia* This is found in asthma in parasitic infections such as ankylostomiasis and hydatid disease in skin diseases such as psoriasis urticaria and dermatitis herpetiformis in dermato myositis and at times in acute polyarteritis nodosa Hodgkin s disease or leukæmia in chronic abdominal tuberculosis or it may occur as a familial condition of unknown etiology Up to 70% of the white cells may be eosinophils A condition of eosinophilia with splenomegaly is described due to syphilis Hodgkin s disease malaria or some un known cause In 1943 Weingarten described what he called a new disease tropical eosinophilia This is met with in India and is charac terised by severe spasmodic bronchitis leucocytosis and an eosinophilia of up to 89% X ray examination shows a diffuse mottling of the lung fields The condition usually responds rapidly to injections of neoarsphenamine Loeffler in 1932 described a somewhat similar

condition an eosinophilia with shadows as shown by X ray examination in the lung fields *The mast cells* may be increased to about 20% in myeloid leukæmia *The large mononuclears* may increase in infections such as malaria syphilis or trypanosomiasis

### Leucopenia

**Definition** A diminution in the number of white cells in the blood

**Etiology** *Physiological leucopenia* This occurs in old age and in starvation *Pathological leucopenia* This may be met with in tuberculosis influenza typhoid fever pernicious anæmia splenic anæmia hypersplenism Hodgkin s disease after exposure to radium in phosphorus and benzol poisoning in aplastic anæmia and in agranulocytic angina

### Thrombocytosis

A temporary increase in the platelet count occurs after a hæmorrhage childbirth and splenectomy It is met with in polycythæmia vera chronic myeloid leukæmia Hodgkin s disease in the remissions of pernicious anæmia and in the anæmia of chronic sepsis tuberculosis carcinoma etc Thrombocythæmia is described on p 549

## ANÆMIA

The classification of the anæmias remains a most difficult problem, owing to the incompleteness of our knowledge of their etiology For clinical purposes anæmias were formerly grouped as primary and secondary The cause of the primary anæmias was unknown whereas secondary anæmias were due to some recognisable agent such as infection or hæmorrhage As in all cases anæmia must result from a deficient formation of red cells or from their excessive destruction this classification becomes more unsatisfactory as our knowledge increases Wintrobe in 1930 grouped some of the anæmias according to the size and hæmoglobin content of the red cells It should be realised that although a red cell may contain less hæmoglobin than normal in proportion to its size (hypochromic anæmia) the proportional amount of hæmoglobin is never increased There is therefore no such condition as hyperchromic anæmia the high colour index in pernicious anæmia being due to large red cells containing a normal percentage of hæmoglobin Wintrobe s classification is as follows 1 *Macrocytic anæmia* as in pernicious anæmia achrestic anæmia the anæmia associated with liver disorders such as cirrhosis passive congestion carcinoma and acute yellow atrophy and with myxœdema the anæmia of sprue tropical macrocytic anæmia the pernicious anæmia of pregnancy the anæmia of chylous diarrhœa and of Diphyllobothrium latum infestation 2 *Normocytic anæmia* as in aplastic anæmia malaria and after an acute hæmorrhage 3 *Simple microcytic anæmia* as in chronic infections and carcinoma 4 *Microcytic hypochromic anæmia* as in chronic hæmorrhage infestation with hookworms simple achlorhydric anæmia the Plummer Vinson syndrome chlorosis and the simple nutritional

anæmia of infants Severe hypochromic anæmia in males is often associated with a neoplasm of the alimentary tract A definite advantage of Wintrobe's classification is that it emphasises the distinction between macrocytic and microcytic hypochromic anæmias This is of clinical importance as if the red cells are saturated with hæmoglobin the macrocytic anæmias are benefited by liver therapy and the hypochromic anæmias by iron Iron is, however, required for macrocytic anæmias if the saturation index is low

*Davidson* in 1932 put forward a classification with an etiological basis This is obviously sound but there are at present gaps in our knowledge which render it incomplete The following classification is that of Davidson with some modifications 1 *Nutritional deficiency anæmias* These may be due to (*a*) Lack of the hæmopoietic principle (see p 513) This occurs as a primary defect (the diet being adequate) in pernicious anæmia and as a secondary defect (*i e* resulting from some recognisable cause) in the anæmia of sprue dysentery and tropical megalocytic anæmia in the pernicious anæmia of pregnancy in Diphyllobothrium latum infestation in carcinoma of the stomach and after gastrectomy In this latter group there is also often an error in iron assimilation There may be a deficiency of storage of the hæmopoietic principle in advanced cirrhosis of the liver (*b*) Lack of the factors required for hæmoglobin formation such as iron and possibly copper calcium thyroxine vitamin C chlorophyll and a salt balance in the food This also may be a primary defect as in the simple achlorhydric anæmia and the Plummer Vinson syndrome where the food intake is often satisfactory or secondary to a recognisable food or endocrine defect as in the simple nutritional anæmia of infants chlorosis chronic gastritis and enteritis *cœliac* disease starvation, prolonged milk feeding as in the treatment of peptic ulcers and in myxœdema and thyrotoxicosis 2 *Post hæmorrhagic anæmias* These occur after acute or chronic hæmorrhage 3 *Hæmolytic anæmias* (*a*) Acute hæmolysis may result from black water fever, malaria paroxysmal hæmoglobinuria septicæmia or toxins such as snake venom It also occurs in Lederer s anæmia (*b*) A persistent hæmolysis as in congenital or acquired acholuric jaundice, sickle cell anæmia Cooley s anæmia Von Jaksch s anæmia and lead poisoning 4 *Anæmias due to inhibition of the bone marrow function* These may be primary as in aplastic anæmia or secondary as in aplastic anæmia due to noxious agents such as X rays radium emanations benzol lead or mercury A leuco erythroblastic anæmia occurs owing to involvement of bone marrow in carcinomatosis myelomatosis in osteosclerosis including the marble bone disease of Albers Schonberg in myelosclerosis in Hodgkin s disease xanthomatosis of bone and in Gaucher s disease Nucleated red cells are found in the blood in numbers greater than would be expected from the anæmia There may be as many as 50 normoblasts for every 100 leucocytes There may also be occasional myelocytes and myeloblasts

By some writers groups 1 and 4 in the above classification are grouped together as the *Dyshæmopoietic anæmias*

## NUTRITIONAL DEFICIENCY ANÆMIAS

### Pernicious Anæmia

### (*Addisonian Anæmia*)

**Definition** A severe macrocytic anæmia characterised by a typical blood and bone marrow picture and if untreated, a progressive course which is often interrupted by remissions

**Etiology** Pernicious anæmia results from the lack of a hæmopoietic principle This hæmopoietic principle is thought to be formed from the interaction of Castle s extrinsic and intrinsic factors The hæmopoietic principle has been isolated from the liver as vitamin $B_{12}$ or cyanocobalamin It can also be prepared from the liquor obtained in the growth of *streptomyces griseus* The extrinsic factor which is vitamin $B_{12}$ is present in protein food In order that it may become hæmatopoietically active it must be bound to the intrinsic factor which is thought to be a mucoprotein contained in the gastric juice Further if vitamin $B_{12}$ is injected parenterally it becomes active because it is bound to a substance in the tissues and it exists in the blood in the bound form This union is destroyed by heat the vitamin $B_{12}$ being thus rendered hæmopoietically inactive The hæmopoietic principle thus formed is carried to the liver and other organs and stored in the liver and to a lesser degree in the kidneys and spleen In its absence pernicious anæmia develops owing to the failure of conversion of megaloblasts into erythroblasts and normoblasts in the bone marrow When these megaloblasts are liberated into the blood stream they are destroyed by the cells of the reticulo endothelial system The iron which is deposited in the spleen liver and kidneys and the excess of bilirubin in the blood are in part due to this hæmolysis and in part to the failure of the bone marrow to utilise these substances in the production of red cells

In pernicious anæmia the $B_{12}$ in the food is not absorbed owing to the absence of the intrinsic factor from the gastric juice Achylia gastrica has been demonstrated in the majority of cases the gastric juice showing no free HCl pepsin or rennin after the intramuscular injection of 1 ml of 0 1% solution of histamine

Sturgis and Isaacs have shown that a principle active in the treat ment of pernicious anæmia is present in dried whole hog s stomach This gastric extract is potent probably because it contains the intrinsic factor which is necessary for the absorption of vitamin $B_{12}$ in the food

*Predisposing causes* 1 Age Usually after 35 years 2 Sex No definite difference 3 A familial incidence is noted in some cases 4 Extensive gastrectomy up to 30% of such cases may develop pernicious anæmia

**Pathology** Post mortem the body is well nourished The skin has a lemon yellow tint the subcutaneous fat is yellow and the muscles are rather dark red The heart is soft owing to fatty degeneration and tabby cat striation is seen under the endocardium and on the musculi papillares and columnæ carneæ The tongue is smooth and may show ulcers on its edges The mucous membrane of the stomach is

atrophied and petechial hæmorrhages may be seen Microscopical examination shows changes in the fundus with atrophy of glands and disappearance of the parietal and chief cells but the glands in the pylorus are well preserved and no changes are seen in Brunner s glands In man, as opposed to the pig *it appears that the intrinsic factor is* formed in the cardia and body of the stomach The liver is slightly enlarged owing to fatty infiltration The spleen is usually enlarged but may be smaller than normal Owing to the deposition of free iron (hæmosiderin) a prussian blue reaction is given by sections of the liver and occasionally by the spleen and kidneys, on applying a 2 to 5% solution of potassium ferrocyanide followed after washing by 5 to 10% hydrochloric acid *The red marrow of the bones is increased* especially in long bones such as the femur Degeneration may be found in the postero lateral columns of the cord

**Clinical Findings** The patient is usually an adult between the ages of 40 and 70 years Very rarely pernicious anæmia occurs in children He complains of progressive weakness increasing pallor with dyspnœa on exertion palpitations and at times anginal pains due to anoxia of the heart muscle In some cases swelling of the feet or ankles may be noted There may be soreness of the tongue, numbness or tingling in the legs and hands vomiting or diarrhœa Failing vision due to optic atrophy may be the first symptom The atrophy is secondary to a retrobulbar neuritis *the lesion being near the chiasma Other* neurological evidence of subacute combined degeneration may be very slight

*On Examination* The nutrition of the patient is usually good The tongue may be very smooth The skin is pale with at times a lemon yellow tint The hair may be prematurely grey There may be cutaneous pigmentation with areas of leucoderma and small petechiæ *may be seen The spleen is enlarged in about 3% of cases* and the liver may be palpable Evidence of postero lateral spinal sclerosis may be found such as patchy anæsthesia of the legs weakness of muscles and an extensor plantar response Cases in which there is tenderness of the calves weakness of the legs tingling numbness and depressed tendon reflexes may be suffering from peripheral neuritis or an early stage of subacute combined degeneration Loss of vibration sense is often the earliest sign of a cord lesion In some instances there is definite jaundice and ascites The blood A typical count during a relapse phase is as follows Red cells 500 000 to 2 500 000 per c mm Hb 12 to 65% C I 1 1 Mean corpuscular Hb 50$\gamma\gamma$ *Mean corpuscular Hb concentration 33% Saturation index 1* The average diameter of the red cells is 8 24$\mu$ (normal 7 2$\mu$) Mean corpuscular volume 150 c$\mu$ Volume index 1 5 The red cells show anisocytosis poikilocytosis megalocytosis polychromasia and punctate basophilia Normoblasts and megaloblasts are present *The platelets are reduced* Reticulocytes are increased to about 2% White cells There is a leucopenia 4 000 to 5 000 per c mm, with relative lymphocytosis up to 50% The polymorphonuclears show a shift to the right in the Arneth count many having a four or five lobed nucleus *There*

are usually some myelocytes present The coagulation time This is prolonged The sedimentation rate This is increased owing to the anæmia alone and not to tissue destruction The serum An indirect van den Bergh reaction is given The bone marrow obtained by sternal puncture in untreated cases shows 25 to 45% of the cells to be megaloblasts and erythroblasts and premature hæmoglobinisation is present The urine and fæces contain an excess of bile pigment During treatment with liver an output of reticulocytes occurs before the number of red cells increases The fractional test meal shows achylia gastrica no free HCl being secreted after injection of histamine The juice also lacks pepsin and the intrinsic factor" Rarely the juice contains acid and pepsin but the intrinsic factor is absent The stomach empties rapidly Blind gastric biopsy using a flexible gastric biopsy tube shows atrophy of the gastric mucosa in pernicious anæmia and in subacute combined degeneration of the cord

**Differential Diagnosis** The diagnosis of pernicious anæmia depends upon the typical blood count with megalocytosis high colour index and the presence of large nucleated red cells and the fractional test meal showing an achylia and absence of the intrinsic factor During the remission phases although nucleated red cells may be absent in the blood film yet the average size of the red cells remains greater than normal In carcinoma of the stomach the blood count may closely resemble that of pernicious anæmia The X ray findings and occult blood test are usually helpful In sprue and infections with the Diphyllobothrium latum the blood picture may be similar to that of pernicious anæmia but achylia is not present In the megalocytic anæmia of pregnancy the gastric juice contains free hydrochloric acid If there is much pigmentation Addison s disease may be suspected The blood count serves to differentiate Postero lateral sclerosis may occur before the development of the typical blood picture of pernicious anæmia Achrestic anæmia (χρησθαι = to use) is described by Wilkinson and Israels The anæmia is megalocytic the patient being unable to use the hæmopoietic principle Free HCl is present in the gastric juice and there are no complications It is resistant to liver therapy but may respond to folic acid mg 10 daily by mouth

**Course and Complications** Remissions are common during which the patient feels and is better and the blood count improves Blood crises may also be noted characterised by the appearance of large numbers of nucleated red cells Some cases pursue a course rapidly fatal in a few days or weeks in others life is prolonged for 2 or 3 years It is doubtful if permanent recovery apart from treatment ever occurs Even after successful treatment the achylia nearly always remains Complications include trophic ulcers on the buttocks and heels pneumonia and nephritis

**Prognosis** The disease is fatal usually within two to three years if no treatment is given The introduction of the liver and stomach extract treatment has revolutionised the outlook and it is now very rare for a patient to die during the acute stage if adequate treatment is given

**Treatment** The introduction of the Murphy Minot liver treatment *constituted an advance of the first magnitude in medicine*

Originally 250 G of raw or lightly cooked liver of the ox calf or pig were ingested daily Subsequently crude liver extracts were employed, and later more powerful concentrations of liver Vitamin $B_{12}$ is now generally given It is available as Cytamen in ampoules containing 50 100 250 or 1 000 micrograms per ml The initial dose is 100 micrograms intramuscularly repeated daily until a remission is obtained A maintenance dose of 50 to 100 micrograms should then be injected every 2 to 4 weeks for an indefinite period The state of the disease should be checked by periodical blood counts

A reticulocyte crisis is to be expected between the fourth and tenth day of treatment The peak of the reticulocyte rise may touch 55% with 1 million red cells a reticulocyte figure of 35% may be expected with 2 million red cells 14% reticulocytes and with 3 million red cells 4% *reticulocytes The number of reticulocytes then falls rapidly by a* crisis as the mature red cells start to increase in the blood

Larger doses of $B_{12}$ are required if there is sepsis if the patient is elderly or if he has arteriosclerosis In some cases thyroid extract and vitamin C are helpful supplements to treatment

In the later stages of the initial treatment there is often iron deficiency as shown by a mean corpuscular Hb concentration of under 30% *Iron should then be given as ferri et ammon cit gr 30 to 45* t i d and iron is also valuable when nervous symptoms are present Acid hydrochlor dil m 30 to 60 syr aurant m 30 aq ad fl oz 1 Fl oz 1 in 8 oz of water with and after meals t i d will in some cases improve the appetite Folic acid must not be used in the treatment of pernicious anæmia owing to the risk of neurological complications developing More crude liver substances such as Hepastab in doses of 6 to 8 ml intramuscularly at intervals of 1 to 4 weeks are not used now so often in the treatment of pernicious anæmia and this applies also to desiccated preparations of hog s stomach such as Pepsac or Gastrosac the dose being 10 G t i d The powder must be given cold on food or in a drink

## The Pernicious Anæmia of Pregnancy

During pregnancy a macrocytic anæmia of a 'pernicious' type may arise as described by Osler It differs from pernicious anæmia in its tendency to spontaneous recovery with the termination of pregnancy and in the presence of free hydrochloric acid in the gastric juice There may be hæmorrhages from the nose alimentary tract or the vagina The deadly pallor dyspnœa tachycardia fever syncopal attacks transient paralysis or even blindness suggest the occurrence of an internal hæmorrhage

*Differential Diagnosis This anæmia must be differentiated from* Addisonian pernicious anæmia complicated by pregnancy from an aplastic or chlorotic (hypochromic) anæmia during pregnancy and from an acute internal hæmorrhage

**Treatment** *An immediate blood transfusion of 500 ml is required*

followed by the administration of vitamin $B_{12}$ as for pernicious anæmia If no response is obtained folic acid mg 20 should be given daily by mouth It is seldom necessary now to terminate pregnancy

## The Anæmias of Sprue and of Diphyllobothrium Latum Infestation, Tropical Megalocytic Anæmia

These anæmias are of the macrocytic type but the gastric secretion is normal The tropical megalocytic anæmia is probably due to a dietary deficiency In Diphyllobothrium latum infestation it is thought that the worm ingests vitamin $B_{12}$ from the intestinal contents so that it is not available for the host

**Treatment** A good response is usually obtained with liver concentrates provided they are not highly purified or with Marmite ½ oz daily Proteolysed liver has been found of value in cases of megaloblastic anæmia refractory to the parenteral administration of liver including idiopathic cases and those associated with pregnancy sprue and the puerperium The dose is 2 to 8 teaspoonfuls of the powder daily dissolved in warm water flavoured with pepper and salt Folic acid in doses of mg 10 to 20 daily by mouth combined with liver is of great value in nutritional macrocytic anæmia

## Simple Achlorhydric Anæmia (Faber)

(*Idiopathic Microcytic Anæmia Essential Hypochromic Anæmia*)

The patient is usually a woman of middle age She complains of pallor dyspnœa swelling of the feet palpitations indigestion and in a severe case of anginal pain The predominance in the female sex is probably due to uterine losses of blood The food intake is often satisfactory but may be deficient in iron or the iron is not adequately absorbed Normally 15 mg of iron per day are required

*On Examination* The skin is pale and often sallow The nails are concave or spoon shaped (koilonychia) in many cases and brittle Cracks are often seen at the corners of the mouth The tongue is smooth red but not sore The spleen may be enlarged There are no changes in the spinal cord The test meal shows a complete achlorhydria with rapid stomach emptying and mucus is present in excess there is usually some pepsin and some HCl response to the intramuscular injection of histamine Castle has shown that if beef is incubated with the gastric juice the hæmatinic principle of pernicious anæmia is produced even in specimens of histamine refractory gastric secretions This shows that the juice contains Castle s intrinsic factor and explains why the patient does not develop pernicious anæmia Occasionally normal test meal findings are recorded The van den Bergh test on the blood is negative the fæces contain no occult blood The blood count is that of a hypochromic microcytic anæmia with a low colour index such as red cells 3 to 4 millions per c mm Hb 30 to 50% C I 0 4 to 0 5 The majority of the red cells are smaller than normal Complications include the Plummer Vinson syndrome

*Treatment* *If possible the cause of the anæmia should be removed*

It is well to give a scale preparation of iron such as Ferri et ammon cit in doses rising from gr 5 to gr 60, aq chloroform ad fl oz 1 Fl oz 1 t d s p c Alternatively tab Fersolate gr 3 one t i d p c may be given In any case sufficient iron must be given to provoke a reticulocyte crisis Ferrous gluconate (Fergon) tablets are said to be less likely to provoke indigestion The dose is 1 to 2 tab t i d before meals Alternatively iron may be given by intravenous injection The total amount of elemental iron required is mg 24 5 for each 1% deficit of hæmoglobin as determined on the Haldane scale plus 50% to restore the iron reserves in the tissues to normal A preparation of saccharated iron oxide such as Ferrivenin may be used injecting mg 50 on the first day mg 100 on the second and third days and mg 200 daily subsequently until the calculated dose is given Great care must be taken that none of the fluid extravasates outside the vein The injection must be made slowly about 2 ml a minute otherwise venospasm may occur The ampoules contain mg 100 per 5 ml The total amount injected should never exceed 1 G for fear of provoking hæmosiderosis and a second course should not be given until 3 months have elapsed Iron may also be injected intramuscularly as an iron dextran compound in the form of Imferon The dose is 2 to 4 ml daily, which is equivalent to 100 to 200 mg of iron until the requisite amount has been injected Brown discoloration of the skin over a large area due to leak of the drug along the needle track is a disfiguring complication Acid hydrochlor dil m 20 to 40 t d s p c in a glass of water should also be given in cases of achlorhydria A blood transfusion may be required in very severe cases

**The Plummer-Vinson Syndrome**

This is described on p 18

**The Nutritional Anæmia of Infancy**

Mackay showed that 51% of artificially fed babies and 45% of breast fed babies in London of the hospital class were anæmic This is due to deficiency of iron and minerals in milk and is curable by the administration of iron The following mixture is used Ferri et ammon cit gr 1½, aq chlorof m 60 Add 2 to 3 drops of the mixture to the feeds three times a day and gradually increase up to m 60 t i d The mother also is frequently anæmic

**Chlorosis**

*(The Green Sickness)*

Chlorosis which was so common in the early part of this century in young women working in shops is practically never seen now The characteristic features were the hypochromic anæmia increased blood volume, excess of gastric hydrochloric acid and a greenish tinge in the skin Now we occasionally see a similar condition in adolescent males and females although the greenish tinge is lacking The disease usually responds well to fresh air correction of constipation a diet containing meat and vegetables and the administration of iron

## POST HÆMORRHAGIC ANÆMIAS

### The Anæmia of Acute Hæmorrhage

**Etiology** The hæmorrhage may be external or internal and due to various causes

**Pathology** The regenerative changes in the blood after a severe hæmorrhage include a polymorphonuclear leucocytosis taking place within a few hours followed by a more gradual dilution of the blood with fluid derived from the connective tissue spaces Reticulocytes (up to 5%) are seen and the red cells show anisocytosis and poly chromasia with an occasional normoblast The colour index is low for several weeks A rapid loss of one third of the total blood volume usually proves fatal

**Clinical Findings** A severe hæmorrhage results in pallor restless ness a feeling of sinking through the bed thirst and faintness The pulse is frequent and its volume small The blood nitrogen may rise and uræmia ensue

**Treatment** A blood transfusion is required in severe cases The recipient should be given sufficient alkali by mouth to render the urine alkaline before the transfusion as this will tend to prevent a tragedy due to hæmolysis and precipitation of acid hæmatin in the renal tubules In other cases iron should be given in large doses such as ferri et ammon cit gr 80 t i d It should be noted that this type of anæmia responds to whole liver but not to liver extract probably on account of the iron in the liver

### The Anæmia of Chronic Hæmorrhage

**Etiology** This anæmia results from repeated small hæmorrhages which may be due to such causes as pulmonary tuberculosis hæmor rhoids carcinoma of the alimentary tract etc

**Clinical Findings** The anæmia is of the microcytic hypochromic type with a low colour index and the average red cell is smaller than normal

**Treatment** This consists in the establishment of measures to stop the bleeding and the administration of iron

## THE HÆMOLYTIC ANÆMIAS

### Acute Hæmolysis

In the hæmolytic anæmias there is an increase in the number of reticulocytes in the blood for a period of time with no corresponding rise in the blood count there is also an excess of bile pigment in the blood With rapid blood destruction there is hæmoglobinuria and excess of urobilinogen in the urine and fæces

The hæmolytic anæmias may be classified as follows —

*Hereditary* These are due to intracorpuscular causes such as changes in the shape size amd thickness of the red cells associated with abnormal forms of hæmoglobin They include congenital acholuric jaundice sickle cell anæmia thalassæmia and paroxysmal nocturnal

hæmoglobinuria *Acquired* The anæmia is due to extracorpuscular causes to substances in the plasma which result in hæmolysis They include —

1 Infections Especially malaria and Cl Welchii septicæmia
2 Chemical substances Lead *phenylhydrazine sulphonamides* arsenic phosphorus quinine and trinitrotoluene
3 Allergy Idiosyncrasy to the bean *vicia faba*
4 Immune reactions Mismatched blood transfusion erythroblastosis fœtalis paroxysmal cold hæmoglobinuria virus pneumonia and idiopathic acquired hæmolytic anæmias of unknown origin including Lederer s anæmia Symptomatic hæmolytic anæmia may be an early manifestation of disseminated lupus erythematosus it may also occur in the reticuloses in lymphosarcoma carcinomatosis rheumatoid arthritis multiple myelomatosis and in chronic leukæmia
5 Hypersplenism This results in a hæmolytic anæmia due to overactivity of the spleen and reticulo endothelial system

## Lederer's Anæmia

### (*Acute Febrile Anæmia*)

**Etiology** The cause is unknown but the presence of hæmolysins in the blood is considered to be the exciting factor in some cases

**Pathology** The spleen may be enlarged with infarction and the bone marrow is hyperplastic with an erythroblastic reaction

**Clinical Findings** The patient is usually an adult under the age of 20 He is suddenly taken ill with malaise headache fever (100 to 104° F ) and often a rigor vomiting and epistaxis The passage of dark urine (hæmoglobinuria) may be the first symptom

*On Examination* The patient may be slightly or definitely jaundiced and the urine may contain blood and bile pigments The spleen may be palpable The blood Red cells 2 millions per c mm or less In children the red cells are usually macrocytic but in adults they may be microcytic normocytic or macrocytic C I usually about 1 Some nucleated red cells may be present The reticulocytes are increased The white cells are usually increased (20 000 to 100 000 per c mm ) and myelocytes and myeloblasts may be present In children a lymphocytosis may occur with up to 10% of lymphoblasts The blood culture is sterile

**Differential Diagnosis** At the onset gastric influenza is often suspected It may be difficult to differentiate the disease from acute leukæmia unless recovery occurs

**Prognosis** Death may rapidly occur or the temperature may fall to normal and the patient recover in a few weeks

**Treatment** An immediate blood transfusion after very careful cross matching should be given and repeated if necessary If there is not a good response to three or four transfusions splenectomy should be performed

## Chronic Hæmolysis

Chronic hæmolytic anæmia occurs in acholuric jaundice in sickle cell anæmia and at times in Hodgkin s disease

**Congenital Acholuric Familial Jaundice**

(*Hæmolytic Icterus Hereditary Spherocytosis Minkowski s Disease*)

**Definition** A disease characterised by enlargement of the spleen slight icterus and increased fragility of the red cells

**Etiology** Experimental evidence suggests that a defect in the spherocytes causes them to be unduly fragile No antibodies are found in the blood by Coombs test and transfused red cells survive normally The spleen may destroy these abnormally thick and unduly fragile red cells There is a familial incidence with an equal sex distribution The liability to the disease is transmitted as a dominant Mendelian character The symptoms are usually noted before the age of 10

**Pathology** The spleen is moderately enlarged but the capsule is not usually thickened The pulp contains many red cells which are ingested and destroyed by the endothelial cells The red bone marrow is hyperplastic

**Clinical Findings** Often no symptoms are noted but the patient may complain of jaundice or of attacks of vomiting or of weakness and anæmia

*On Examination* There is some icterus of the skin and conjunctivæ The spleen is enlarged Long standing ulceration of the legs may be noted The urine is dark and contains urobilin but no bilirubin The blood The red cells are fragile undergoing hæmolysis in 0 75% NaCl solution whereas normally hæmolysis does not occur until the strength of the NaCl is lowered to 0 45% The average diameter of the red cells is reduced (microcytosis) There is some anæmia but the colour index is generally just under unity A few normoblasts and an excess of reticulocytes are present The white cells are normal Blood crises occur in which there is a more marked anæmia a leuco cytosis and the number of reticulocytes is increased The crises may be due to increased hæmolysis or to aplastic changes in the bone marrow The serum gives an indirect van den Bergh reaction the jaundice being of a hæmolytic type The fæces are dark and contain stercobilinogen and stercobilin Other congenital defects such as oxycephaly may be present

**Differential Diagnosis** This is established by the familial incidence, the microcytosis with jaundice and the increased fragility of the red cells A blood examination during a crisis may suggest a leukæmia or the acute hæmolytic anæmia of Lederer owing to the leucocytosis with the presence of some primitive granulocytes

**Course and Complications** The disease does not necessarily produce any serious effects upon the patient s health but an intercurrent septic infection may result in an anæmic breakdown later in life Gall stones and deposits of urates around the joints may occur subsequently

**Prognosis** This is good with adequate treatment.

**Treatment** Splenectomy will cure the condition as regards the jaundice and anæmia although the fragility of the red cells after splenectomy is still greater than normal It is only required in cases in which crises occur and should be performed in the remission stage

and a blood transfusion should only be given after careful cross matching In very severe crises splenectomy may be performed as an emergency measure after a small preliminary blood transfusion

### Acquired Acholuric Jaundice

*(Acquired Hæmolytic Icterus)*

**Definition** A variety of acholuric jaundice appearing in adults

**Etiology** The cause is usually unknown but *in some cases the* anæmia is associated with previous blood transfusions Some authorities consider the disease is the same as congenital acholuric jaundice differing only in quantitative rather than qualitative blood reactions Symptomatic hæmolytic anæmia is also described occurring in various infections such as tuberculosis malaria and syphilis or in diseases such as carcinoma cirrhosis of the liver, leukæmia an ovarian tumour or a dermoid Hypersplenism is another causative factor A hæmolysin is present in acquired hæmolytic icterus as transfused red cells are rapidly destroyed The Coombs test is positive indicating a hæmolysin adsorbed to the red cells

**Clinical Findings** The patient is an adult often of the female sex The onset is frequently sudden with nausea vomiting and jaundice Later, attacks of biliary colic may occur

*On Examination* The spleen is enlarged The blood There is a hæmolytic anæmia, but the blood changes are inconstant Spherocytosis and increased fragility may be present The red cells may be *normocytic* or macrocytic The reticulocytes are usually increased The indirect van den Bergh test is positive and excess of urobilinogen is found in the fæces Hæmolytic crises may occur The urine contains urobilin Gall stones occur as a complication

**Treatment** Splenectomy will often effect a cure but should not be performed until any possible cause of the disease has been treated Blood transfusion is usually required before the operation but it must only be given after very careful cross matching followed by the Coombs test for sensitisation After splenectomy the Coombs test usually remains positive

### Sickle cell Anæmia

*(Drepanocytic Anæmia)*

**Definition** A severe anæmia in which the red cells, when deprived *of oxygen assume an elongated or sickle shape*

**Etiology** The cause is unknown but it is thought to be a developmental abnormality of the red cell Sickle cell anæmia occurs chiefly in negroes of West Africa and North America Both sexes are affected and there is a familial incidence the sickle cell trait being transmitted as a Mendelian dominant The patient is usually under 30 years of age

**Pathology** The liver is usually enlarged and there is *hypertrophy* of bone marrow and lymphoid tissue The spleen is enlarged early in this disease owing to congestion but later as the result of siderofibrosis

it is small The sickling depends upon the partial oxygen pressure The deformity of the red cell is due to the hæmoglobin not remaining in solution The hæmoglobin is abnormal and is known as hæmoglobin S Abnormal hæmoglobin C and D are found in some cases If the arm or finger is compressed in order to reduce the oxygen supply imme diately before the blood is taken the number of sickle cells seen in the direct film may be 90% The sickle cell trait results from an individual receiving hæmoglobin S from one parent only (heterozygous state) If he receives it from both parents (homozygous state) he will suffer from sickle cell anæmia

**Clinical Findings** In some cases there are no symptoms the patient having only the sickle cell trait in which the red cells become sickle shaped when incubated under a cover slip for longer than 24 hours This is much more common than sickle cell anæmia In others when both parents have the sickle cell trait the disease manifests itself by acute attacks in which the patient complains of weakness pains in the muscles and abdomen vomiting diarrhœa irregular fever and at times ulceration of the legs Slight jaundice may be present The blood The red cells are diminished in number The average size is slightly enlarged (Wintrobe) The sickle shape appearance of the red cells is only seen in blood which is freshly drawn and allowed to stand under a sealed cover slip In about 12 to 24 hours over 60% of the red cells become elongated and return to their normal shape in another 24 to 48 hours There is anæmia of varying degree with some nucleated red cells polychromasia and reticulocytosis The colour index is below unity There is a leucocytosis of 12 000 per c mm or more The fragility of the red cells is decreased and the indirect van den Bergh reaction is positive If sickle cells are transfused into a normal person they are quickly destroyed but if normal cells are transfused into patients suffering from a sickle cell anæmia they survive normally

**Prognosis** Recovery rarely takes place the patient usually developing an intercurrent infection before the age of 35

**Treatment** General hygienic measures should be adopted and a course of liver and iron treatment tried

## Cooley's Anæmia

(*Thalassæmia*)

**Definition** The major form is a severe anæmia occurring in child hood with splenomegaly and characteristic bone changes A minor form is described as familial microcytic anæmia

**Etiology** The cause is unknown It occurs chiefly in the Medi terranean areas rarely in England and America There is a familial incidence transmission being as a Mendelian dominant When both parents have the minor trait the affected children develop the severe anæmia

**Pathology** There is hyperplasia of the bone marrow with islands of megaloblasts The spleen is fibrosed

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**Clinical Findings** The patient is an infant or child who suffers from lassitude The skin is yellowish or muddy in colour the aspect somewhat Mongoloid with thickening of the malar bones The spleen and liver are enlarged The blood The serum iron is raised The anæmia is microcytic and hypochromic and target cells are seen in which the hæmoglobin forms a circular area in the centre of an otherwise colourless corpuscle Red cells 2 to 4 millions per c mm There is anisocytosis poikilocytosis and many normoblasts Hb 30 to 60% C I low Fragility normal White cells 15 000 to 50 000 per c mm A few myeloid cells are seen in severe cases Platelets normal Indirect van den Bergh positive X ray examination shows changes in the skull the long bones and the small bones of the hands and feet There is rarefaction and trabeculation of the long bones Radiating spicules are seen in the lateral view of the skull

**Differential Diagnosis** Cooley s anæmia is closely allied to von Jaksch's anæmia Other conditions which require exclusion are acholuric jaundice sickle cell anæmia leukæmia and syphilis

**Course and Complications** The disease is steadily progressive

**Prognosis** Death usually occurs before the age of 10

**Treatment** The administration of iron is not usually of any value Splenectomy is occasionally advised

### Von Jaksch's Anæmia

*(Splenic Anæmia of Infants Anæmia Pseudo leukæmia Infantum)*

A disease of infants characterised by enlargement of the spleen liver and lymph glands a severe anæmia and leucocytosis It was originally described in 1889 but later it was considered that the condition described by Von Jaksch might be caused by anæmia associated with many conditions such as rickets under nutrition syphilis congenital abnormalities of the red cells and possibly an early stage of leukæmia The modern tendency is therefore to discard this syndrome as not being a clinical entity

## ANÆMIAS DUE TO INHIBITION OF BONE MARROW FUNCTION

### Aplastic Anæmia

**Definition** A severe anæmia closely resembling pernicious anæmia but the course is not interrupted by remissions

**Etiology** Aplastic anæmia is classified as a primary anæmia as in many cases no cause can be discovered In some cases it is due to exposure to atomic bomb explosion to X rays or radium to carbon tetrachloride or to poisoning with benzol gold phenylhydrazine the sulphonamides or mustard gas It may also occur as a terminal phase of untreated pernicious anæmia or of polycythæmia rubra or be due to replacement of bone marrow by sarcomatous or carcinomatous tumours or it may complicate acute infections especially in children such as influenza and diphtheria

**Pathology** There is marked aplasia of the marrow of the long

bones the normal marrow being replaced by fat and iron is not deposited in the internal organs

**Clinical Findings** The patient is usually a young adult of either sex who notices weakness pallor dyspnœa and palpitations as in pernicious anæmia The skin may have a yellow tinge Purpura or hæmorrhages from mucous membranes or from the uterus may occur The blood Red cells 0 5 to 1 5 million per c mm The cells are generally of normal appearance There are usually no reticulocytes Hb 10 to 30% C I 0 9 to 1 White cells 1 000 to 2 000 per c mm There is a relative lymphocytosis (up to 60%) No myelocytes are seen Platelets are diminished Bone marrow removed by sternal trephine is usually hypoplastic showing a lack of mature cells occasionally it is hyperplastic with many primitive cells Achlorhydria is not present The van den Bergh reaction is negative

**Differential Diagnosis** The severe nature of the anæmia is apparent The blood count distinguishes it from pernicious anæmia and agranulocytosis In aleukæmic leukæmia lymphoblasts or myeloblasts are present and examination of the bone marrow serves to differentiate

**Course and Complications** The course is steadily progressive

**Prognosis** Death is to be expected in less than a year unless there is some cause which can be removed

**Treatment** The effect of liver treatment in this disease is usually disappointing Iron and arsenic are without avail A blood transfusion produces only temporary improvement but repeated drip transfusions may keep the patient alive for several years Concentrated suspensions of fresh red cells are preferable to whole blood as they are required less frequently When the hæmoglobin falls to 55% a transfusion should be given sufficient to raise the hæmoglobin to over 80% Very occasionally splenectomy is successful more often the patient dies within a few days of the operation

## Agranulocytosis

(*Agranulocytic angina Granulocytopenia Malignant neutropenia*)

**Definition** A disease characterised by a marked diminution in the number of granulocytes in the blood (neutropenia) with ulceration in the mouth rectum or vagina

**Etiology** A few cases are of the acute idiopathic type associated with idiopathic aplastic anæmia The majority are due to substances which poison the bone marrow Amidopyrine and some sulphonamides are particularly dangerous and a small dose of either may sensitise the patient Noxious substances thus include amidopyrine (Pyramidon) Amidophen Gardan Novalgin or barbiturates containing amidopyrine such as Allonal Cibalgin Compral Somnosal Veramon and Veropyron Dinitrophenol used for slimming benzol bismuth and gold salts thiouracil atomic bomb radiations neoarsphenamine carbimazole Butazolidin Tridione Diamox chlorpromazine and chloramphenicol may also cause agranulocytosis Very severe infections may also cause agranulocytosis These include osteomyelitis sinusitis lateral sinus thrombosis septicæmia and liver abscess It is possible that in

cases associated with infection agranulocytosis is the primary change and this allows the infection to develop unchecked Hypersplenism may also cause agranulocytosis, possibly through an exaggerated inhibitory effect of a splenic hormone on the bone marrow, possibly because of overdestruction of white cells by the spleen Agranulocytosis may result from irradiation and from the use of radioactive *phosphorus, or be a part of the blood picture in aplastic anæmia or* aleukæmic leukæmia *Predisposing causes* 1 Age Usually adults 2 Sex Females predominate

**Pathology** There is a marked diminution or absence of myeloid cells (aplastic type) or of granulocytes (maturation type) in the bone marrow No leucocidal substance is present in the blood

**Clinical Findings** The disease may have an acute onset with sore throat, dysphagia, headache, shivering generalised myalgic pains and fever In other cases the disease begins insidiously during gold or sulphonamide treatment *Chronic or recurrent cases* also occur in which the white cells from time to time fall below 4 000 per c mm owing to a reduction of granulocytes with accompanying symptoms of ill health and a liability to septic infections

*On Examination* The temperature is raised to about 102° F Ulceration is seen in the mouth, in such sites as the tonsils pharynx or tongue The cervical glands are in some cases enlarged and the spleen *may be just palpable There may be jaundice and necrotic* lesions in the skin, rectum or vagina There is no tendency to bleeding from mucous membranes Meningitic symptoms may occur, the cerebrospinal fluid showing a paretic type of Lange reaction The urine A trace of albumin is usually present The blood There is a leucopenia due to the marked diminution of the granulocytes A typical count is as follows White cells 1 000 per c mm Polymorphonuclears *4%, mononuclears 16% lymphocytes 80%* No immature white cells are seen The red cell and platelet count are normal, or there may be a slight anæmia

**Differential Diagnosis** The throat lesion may suggest Vincent's angina or diphtheria These are excluded by examination of the throat swabs and by the blood count The leucopenia with relative lymphocytosis is differentiated from aleukæmic lymphæmia by the absence of immature white cells Monocytic angina somewhat resembles agranulocytosis In the former the white cells show a high proportion of monocytes (up to 80%) In aplastic anæmia the red cell count is low

**Course and Complications** In acute untreated cases the course is usually rapidly progressive to a fatal issue Complications include bronchopneumonia and jaundice

**Prognosis** The prognosis has been much improved by the use of penicillin except in the acute idiopathic type and in severe secondary cases The response of the maturation type is more favourable than that of the aplastic type

**Treatment** *Prophylactic* Amidopyrine is a dangerous drug and should not be prescribed There appears to be no justifiable reason *for the use of gold salts* Sulphonamides should not be given for longer

than 7 to 10 days and drug fever is an indication for their immediate discontinuance

*Curative* No definite curative treatment is known A line of treatment which has been successful in some cases is a course of penicillin This is based on the hope that if the risk of secondary infection which is usually the cause of death in agranulocytosis is removed or minimised by the penicillin the bone marrow will recover its power of forming granulocytes and once the noxious agent is removed a cure will result 300 000 units of a procaine penicillin should be injected every 12 hours for about a week and if there are mouth lesions a penicillin lozenge may be sucked every hour for 8 doses Achromycin (tetracycline) should be given by mouth mg 250 capsule 4 times daily Vitalin $B_6$ (pyridoxine) may also be helpful and 200 mg should be injected intravenously daily for a week Fluids up to 5 pints should be taken daily Pentnucleotide is not often used now The local necrotic areas should be appropriately treated Thus the mouth should be sprayed before feeding with Nupercaine (cinchocain hydrochlorid B P ) 1% in glycerin 75% and water 24% if pain prevents swallowing Penicillin lozenges may be taken as described above or the mouth swabbed after feeds with hydrogen peroxide (10 vols ) diluted with an equal quantity of water followed by a swabbing with a citric acid solution of gr 10 to 1 fl oz In cases of hyperplenism splenectomy is usually advisable

## THE LEUKÆMIAS

**Definition** Progressive diseases of the hæmopoietic system characterised by an increase in the white cells and their precursors in the blood and changes in the myeloid or lymphoid tissues of the body

**Etiology** It is probable that the changes are of the nature of a new growth and result in some cases from exposure to X rays and to radiations from atomic bomb explosions

*Varieties* Leukæmia may be acute or chronic and is subdivided further into acute lymphatic leukæmia (lymphadenosis) acute myeloid leukæmia (myelosis) acute monocytic (histiocytic) leukæmia chloroma chronic lymphatic leukæmia and chronic myeloid leukæmia

**Pathology** The types of white cells which are seen in the blood in the leukæmias are largely embryonic such as myelocytes myeloblasts lymphoblasts etc The only way to learn to recognise these cells is by having them demonstrated in blood films under the microscope and a detailed description will not therefore be given (see Fig 46)

### Acute Lymphatic Acute Myeloid and Acute Monocytic Leukæmias

Clinically it is impossible to distinguish between these diseases apart from the blood examinations They are comparatively rare

**Pathology** *Acute Lymphatic Leukæmia* There is hyperplasia of lymphatic tissue throughout the body with enlargement of the lymph glands and infiltration of the spleen liver and bone marrow with lymphocytic cells

*Acute Myeloid Leukæmia* The lymph glands are enlarged to a

varying degree, and the spleen, liver and bone marrow are infiltrated with myelocytes and premyelocytes

*Acute Monocytic Leukæmia* The spleen liver bone marrow and lymph glands are infiltrated with embryonic monocytes (histiocytes)

In all varieties hæmorrhages may be found in such organs as the stomach intestines, lungs brain, kidneys into the buccal mucous membrane and under the skin

**Clinical Findings** The patient is usually a male under the age of 20 Females are only affected half as frequently The onset usually resembles a feverish attack or chill, or a hæmorrhage from the mouth rectum vagina or other site may be the initial symptom Intractable hæmorrhage after dental extraction may be the first indication of the disease The patient may complain of pain in the bones

*On Examination* The patient looks pale and ill, and purpuric spots or larger ecchymoses may be seen in the skin The temperature is irregular between 99° F (m) and 101 F (e) or higher The pulse is proportionately rapid Necrotic lesions may be seen in the mouth or fauces The liver or spleen may be just palpable and the glands in the neck, axillæ or groins enlarged In acute myeloid leukæmia there may be tenderness on tapping over the sternum A mediastinal type occurs (see Fig 47) in which pressure signs point to a mediastinal tumour this is confirmed by X ray examination The blood count (*a*) *Acute lymphatic leukæmia* Red cells 3 millions per c mm or less Hb 50% or less C I 0 8 to 0 9 There may be anisocytosis with a few megaloblasts or normoblasts Platelets usually reduced White cells 10 000 to 100 000 per c mm The majority of cells are non granular Polymorphonuclears 2%, lymphocytes—some with smeared nuclei and lymphoblasts 98% The lymphocytes may be small or large The oxidase reaction is negative as the cells are agranular (*b*) *Acute myeloid leukæmia* Red cells as above but often megalocytes and some megaloblasts are seen White cells 20 000 to 200 000 per c mm Polymorphonuclears 2 to 4% Lymphocytes 3% Myeloblasts and myelocytes 93% The myeloblastic cells give the oxidase reaction as they are granular (*c*) *Acute monocytic leukæmia* Red cells as above White cells 50 000 per c mm Monocytes 87% Polymorphonuclears 4% Lymphocytes 9% Later a few monoblasts may be found In some cases the blood changes are aleukæmic in type Sternal puncture shows the bone marrow very cellular 70 to 99% of the cells being primitive white cells of either the myeloid or lymphatic series

**Differential Diagnosis** The onset of the illness may suggest a feverish cold or influenza Hæmorrhage may point to a lesion of the lungs, stomach or intestines The enlarged glands fever and palpable spleen or liver occur also in glandular fever The continuous fever may arouse suspicions of infective endocarditis tuberculosis or enteric fever The hæmorrhages in the skin or mucous membranes might be due to purpura or scurvy The diagnosis is established by the blood count and examination of the bone marrow attention should be paid to the changes also present in the red cells which are a distinctive feature of acute leukæmia as opposed to glandular fever



FIG 47 DILATED VEINS ON TRUNK AND ARMS RESULTING FROM OBSTRUCTION OF THE SUPERIOR VENA CAVA

The patient aged 12 was suffering from subacute lymphatic leukæmia with a mediastinal leukæmic tumour The first symptom was intermittent swelling of the face and neck which disappeared as the collateral circulation was established

**Course and Complications** The course usually is rapid, but spontaneous remissions may occur Subacute cases occur characterised by the bone marrow findings of acute leukæmia the formation of tumours in the mediastinum abdomen or bones (see chloroma) and in which there is little or no fever hæmorrhages are unusual and the fatal issue may be postponed for about six months Complications include the hæmorrhages into various sites and *leukæmic retinitis*

**Prognosis** The outlook is hopeless, death usually occurs in a few days to a few weeks from the onset A cerebral hæmorrhage may cause rapid death

**Treatment** Treatment both by aminopterin and by urethane has proved disappointing In the undifferentiated acute leukæmia of children an initial remission lasting from 3 weeks to 3 months may be obtained in about 85% of cases by the administration of cortisone It can be given by mouth in doses varying from mg 25 to 100 every 6 hours Necrotic mouth lesions should be treated as described under agranulocytosis X ray treatment may cause a temporary disappearance or diminution of a mediastinal mass

## Chloroma

**Definition** A variety of subacute myeloid or rarely lymphatic leukæmia accompanied by the formation of tumours in the subperiosteal tissues and elsewhere

**Etiology** The cause is unknown Chloroma occurs usually in male children

**Pathology** Greenish nodules of lymphoid tissue are found in all the organs of the body except the brain and they occur under the periosteum and in the bone marrow especially in the orbit skull long *bones vertebræ ribs and sternum The green colour the cause of* which is unknown fades on exposure to air

**Clinical Findings** The symptoms may resemble those of acute leukæmia Early nervous symptoms such as pains in the back and legs and inability to walk may be the first indication of ill health In addition an orbital tumour may cause protrusion of the eye and swellings in the skull may result in facial paralysis deafness or blindness A sternal swelling may be apparent The blood count resembles that of acute myeloid or rarely lymphatic leukæmia

**Prognosis** *Death occurs rapidly usually in 4 to 8 months from* diagnosis

**Treatment** There is no cure and treatment is only symptomatic

## Chronic Lymphatic Leukæmia

**Pathology** The lymph glands are enlarged and on section show an excess of lymphoid cells The spleen is enlarged with lymphatic infiltration The liver is enlarged the periportal connective tissue is infiltrated with lymphocytes The bone marrow of the long bones is *grey and shows lymphoid metaplasia*

**Clinical Findings** The patient is usually a male of middle age He complains of weakness pruritus bone pains swelling of the tonsils

enlargement of the glands in the neck or elsewhere, or of swelling of the abdomen caused by the spleen

*On Examination* Groups of enlarged glands may be seen and felt in the neck, axillæ elbows and groins They are soft early in the disease but later are hard they are not tender The skin is freely movable over them In some instances there is a generalised cutaneous lesion resembling exfoliative dermatitis and small nodules may be felt in the skin (leukæmia cutis) The skin in certain areas may be slightly red and thickened owing to lymphatic hyperplasia The tonsils may be enlarged owing to lymphatic infiltration The spleen and liver are usually enlarged often to a considerable degree There is frequently a slight but irregular fever Cranial nerve palsy has been noted in some cases the VI and VII nerves being especially liable to be affected The blood count Red cells 3 millions per c mm or less Hb 60% or less C I 0 6 White cells about 90,000 per c mm Polymorphonuclears 2 to 8% Small lymphocytes 92% Large lymphocytes 2% An occasional lymphoblast The bone marrow obtained by sternal puncture shows 40 to 90% of the cells to be lymphocytes The basal metabolic rate is raised

**Differential Diagnosis** The disease must be differentiated from other causes of chronic glandular and splenic enlargement, such as myeloid leukæmia Hodgkin s disease tuberculosis syphilis etc Enlargement of the mediastinal glands may produce cough simulating whooping cough The blood count establishes the diagnosis There is however a variety of chronic leukæmia in which the total number of white cells is not increased but the differential count is similar to that given above This is called *aleukæmic lymphæmia* Examination of the bone marrow is of great help in establishing the diagnosis

**Course and Complications** The disease usually pursues a chronic course of several years duration The number of large lymphocytes and lymphoblasts in the blood often increases indicating the probability of an acute termination The presence of myelocytes may be indicative of bone marrow stimulation Complications include Venous thrombosis and rarely hæmorrhages as in the middle ear retina or stomach

**Prognosis** Death usually occurs in from 3 to 5 years from the date of diagnosis

**Treatment** When there is lymphocytosis but the lymph glands are not markedly enlarged and there is little anæmia special methods of treatment are best avoided X rays are usually effective in reducing the size of lymph gland masses but the total number of white cells in the blood is seldom reduced unless X ray treatment is given to the spleen Blood transfusion is helpful in cases of anæmia Radioactive phosphorus ($P^{32}$) can also be given The dose is 6 millicuries intravenously repeated in 6 or 8 weeks if necessary It does not produce radiation sickness The initial dose by mouth of $P^{3}$ is 2 millicuries followed by 1 millicurie once or twice a week for *4 to 5 doses* and subsequently every week or two weeks until the white cell count falls to about 30 000 (see also p 541) Arsenic is now seldom used but urethane is occasionally temporarily *successful* as an alternative to, or in addition to X ray treatment It is given by mouth as Urethane

gr 15 (1 G) syr aurant m 25 aq chlorof ad fl oz ¼ 1 G of urethane may be given t d s and the total amount required to lower the white cell count to about 20 000 per c mm varies from 19 G to 134 G the average course lasting 30 days Chlorambucil (γ p (N N di (2 chloro ethyl) amino phenylbutyric acid)) has been favourably reported on The dose is 0 1 to 0 2 mg /kg body weight by mouth for 3 to 6 weeks When hæmolysis occurs cortisone or ACTH may produce a temporary improvement Splenectomy is sometimes recommended if there is great pain and discomfort from the enlarged spleen or if there is severe hæmolysis and thrombocytopenia

### Chronic Myeloid Leukæmia

*(Chronic Granulocytic Leukæmia)*

**Pathology** The clotted blood may have a greenish white colour The lymph glands These are only slightly affected The mesenteric glands may be enlarged The spleen is much enlarged and may weigh as much as 18 lbs (normal weight is 5 to 6 oz) There is perisplenitis It is firm on section and whitish nodules are studded in its texture There is myeloid metaplasia of the spleen substance and atrophy of the Malpighian corpuscles The liver is enlarged and areas resembling multiple abscesses are seen consisting of myeloid metaplasia around the intralobular capillaries Portions of the ileum may be infiltrated with myeloblasts resulting in ulceration and perforation The bone marrow This is greyish red and shows proliferation especially of myeloblasts The kidneys show leukæmic infiltration

**Clinical Findings** The patient is usually a male between the ages of 20 and 45 years The onset is insidious with weakness pallor and dyspnœa pain in the region of the spleen abdominal swelling or rarely priapism due to venous thrombosis may be the first symptom noted

*On Examination* The patient is pale The spleen is enlarged at times only just palpable but in other cases filling nearly half the abdominal cavity and friction may be heard over it The liver is enlarged and palpable Usually the lymph glands cannot be felt There may be ascites or œdema of the legs Occasionally small subcutaneous nodules of myeloid tissue are present and in the later stages there are petechial or purpuric lesions of the skin In advanced cases there is marked tenderness of the bones especially of the lower part of the sternum and long bones The urine often contains excess of uric acid owing to the breakdown of leucocytes It may be found that the temperature is slightly raised The basal metabolic rate is raised The blood The uric acid may be increased The blood count Red cells 3 to 5 millions per c mm or less Hb 50% or less C I 0 7 to 1 The red cells may show anisocytosis polychromasia and a few normoblasts or megaloblasts The platelets are usually first increased and later diminished The white cells 300 000 to 500 000 per c mm Differential count Polymorphonuclears 40% Eosinophils 5% Basophils 2% Small lymphocytes 10% Large lymphocytes 5% Large mononuclears 1% Myelocytes 34% (usually neutrophil a few eosinophil and basophil) Premyelocytes

and myeloblasts 8% In *eosinophilic leukæmia* the leucocytes are chiefly mature eosinophil cells with larger granules than are present in normal eosinophils usually eosinophil myelocytes up to 2% are present The bone marrow obtained by sternal puncture shows an increase in myeloblasts premyelocytes and myelocytes *Aleukæmic leukæmia* This resembles *aleukæmic lymphæmia mentioned on* p 530, but in this condition the abnormal white cells are of the granular type (myeloblasts and myelocytes) although the total white cell count is not increased The diagnosis can be established by examination of the bone marrow

**Differential Diagnosis** Myeloid leukæmia must be differentiated from other causes of anæmia especially pernicious anæmia and from other conditions associated with enlargement of the spleen Examination of the blood establishes the diagnosis

**Course and Complications** The course is usually prolonged for 4 or 5 years or more Crises may occur, in which the patient becomes more ill with an increase in the myeloblasts Towards the end the disease may pursue a very rapid course Remissions do not occur spontaneously but may result from secondary infections or from treat ment Complications include Venous thrombosis hæmorrhages in internal organs and muscles labyrinthine involvement causing deafness and vertigo and leukæmic retinitis

**Prognosis** The disease is fatal

**Treatment** Various forms of treatment may be given These include irradiation either by X rays or by radioactive phosphorus ($P^{32}$) The latter does not cause radiation sickness and is about as effective as X rays Nitrogen mustard is given intravenously diluted with saline 0 1 mg /kg body weight for 4 days It is liable to cause vomiting and rise of temperature Busulphan (Myleran) (1 4 di methanesulphonyl oxybutane) has the advantage that it is given by mouth in tablets of mg 2 or mg 0 5 There are no side effects It reduces the size of the spleen and liver lowers the white cell count raises the hæmoglobin level and relieves the patient of his symptoms The standard dose is 0 06 mg /kg body weight daily, which is about mg 4 for an adult The treatment usually lasts for 3 to 7 months as determined by the blood count A second course is given when the symptoms recur usually after an interval of 5 to 18 months It is not believed to be effective indefinitely

## Hodgkin's Disease

(*Lymphadenoma Lymphogranuloma*)

**Definition** A fatal disease characterised by enlargement of lymph glands with the formation of lymphogranulomatous tissue in the spleen and elsewhere

**Etiology** It is usually considered that Hodgkin s disease is either an infective granuloma of unknown etiology or a malignant neoplasm It seems more probable that it should be grouped with the reticuloses in the borderland between a granuloma and a malignant neoplasm Thomson in 1955 suggested it is a form of tumour of the thymus gland

**Pathology** The lymphatic glands and spleen are chiefly involved but in some cases no enlargement of these structures can be seen at autopsy and the diagnosis can only be established by finding lymph adenomatous tissue in the liver and other organs on microscopical examination The glands These are enlarged in different parts of the body such as the neck, mediastinum axillæ abdomen and groins They are firm and greyish white on section although fatty degeneration and tuberculous caseation may occur Microscopically the fibrous tissue is increased there is an infiltration of lymphocytes and peculiar lymphadenoma or giant cells (Dorothy Reed cells) with central nuclei are present The endothelial or epitheloid cells are increased and often there are masses of eosinophil cells The spleen This is mode rately enlarged and firm On section there are white areas of lympho granulomatous tissue ( hard bake spleen) The liver may also be enlarged and contain lymphogranulomatous tissue The kidneys may be similarly affected and very rarely the bladder is involved The lungs may be studded with small nodules The vertebral canal Lymphogranulomatous tissue may spread from the periosteum into the vertebral canal and compress the cord or spinal nerve roots The bodies of the vertebræ may be involved The stomach and intestines may show hyperplasia of lymphatic tissue In some cases the disease appears to start in the reticulo endothelial cells of the thymus The bone marrow may be infiltrated with lymphogranulomatous tissue

**Clinical Findings** The patient is usually a male between 15 and 45 years of age Females are affected only half as frequently The onset is generally insidious the first symptom being usually enlargement of glands in the neck or axilla In other cases weakness or dyspnœa may first attract attention Further symptoms depend upon the site of the enlarged glands or of the lymphogranulomatous infiltration Thus pain may result from pressure on nerve trunks going to the extremities or chest intense dyspnœa may be caused by tracheal or bronchial constriction there may be cutaneous irritation or diarrhœa from intestinal involvement Frequency of micturition and dysuria are symptoms of bladder involvement In latent cases a persistent irregular or intermittent fever may be an early symptom Alcohol sometimes causes severe pain at the site of one of the lesions This may be the first symptom of the disease

*On Examination* There is usually pallor and the enlarged glands may be seen projecting from the side of the neck or forming a collar The glands may also be visible or palpable in the axillæ or groins The glands are firm slightly mobile and the skin over them is neither adherent nor discoloured Examination of the chest may reveal signs of mediastinal glandular enlargement or weak air entry over the apex of one lung unequal pupils or a pleural effusion Dilated venous radicles may be seen over the upper part of the chest back or front The spleen is usually moderately enlarged the liver just palpable and ascites may be present or jaundice due to pressure of glands in the portal fissure Enlarged abdominal glands cannot usually be felt Pressure on the cord or spinal roots may result in sensory changes in the extremities

or some degree of weakness or paraplegia The skin is usually pale but pigmentation may be seen apart from that resulting from the therapeutic use of arsenic, especially if the abdominal glands are involved Other cutaneous lesions include erythemata, generalised exfoliative dermatitis and rarely small nodules of lymphogranulomatous tissue may be felt under the skin Ulceration may occur in the skin lesions Herpes zoster is usually associated with arsenical treatment but it may occur apart from this and be generalised

*The Relapsing Type* (Pel Ebstein Syndrome) As mentioned above there is usually slight irregularity of temperature in all cases of Hodgkin s disease, but periodical waves may occur with apyrexial intervals The pyrexial periods usually last for 5 or 6 days and recur at intervals of 15 to 86 days the span which is the distance between the crests of the successive waves being fairly constant in each case During the fever the patient feels more ill may vomit and the spleen and glands often enlarge In the intervals the patient feels comparatively well and may gain weight The blood There is no characteristic change In some cases there is a leucocytosis of 20 000 per c mm or more even in the afebrile periods with an excess of polymorphonuclears reduction of lymphocytes and slight excess of monocytes In other cases there is leucopenia In acute cases the number of eosinophils may be much increased and they have large granules When the bone marrow is involved myelocytes and myeloblasts appear in the circulation The red cell count may rarely show a hæmolytic anæmia but in some cases a macrocytic anæmia has been described closely resembling pernicious anæmia but with a normal or increased fragility of the red cells

**Differential Diagnosis** This involves a consideration of other causes of glandular and splenic enlargement of mediastinal pressure of ascites and jaundice and of irregular or periodical pyrexia Enlarged glands may result from tuberculosis leukæmia lymphosarcoma syphilis, sepsis and glandular fever Mediastinal pressure may be due to a new growth aneurysm or enlarged glands from several causes Unexplained pyrexia may be due to tuberculosis infective endocarditis enterica group infections brucella infections malaria etc In all cases the blood must be examined and if possible a superficial gland should be removed and examined microscopically Often however, the pathological report is indefinite

**Course and Complications** Certain types are described according to the course pursued 1 Chronic Hodgkin s disease Here the disease is progressive but interrupted by remissions Life may be prolonged for four or five years 2 The relapsing type Described above 3 The acute type (Hodgkin s sarcoma) in which the disease is rapidly fatal Complications result from mediastinal pressure intercurrent infection amyloid degeneration or tuberculous infection

**Prognosis** The disease is invariably fatal although a remission of several years may occur following X ray treatment in early cases

**Treatment** There is no known cure Adequate rest fresh air and nourishing food should be ensured Arsenic usually causes a temporary improvement X ray treatment is still the standard form

of treatment It usually causes a temporary remission the glands diminishing in size or disappearing With relapse it is usual for other glands to swell and finally no response is obtained and the patient dies Treatment with radioactive metals such as phosphorus has not met with more success than radiotherapy possibly because so much radioactive phosphorus would have to be given as to cause bone marrow damage Nitrogen mustard may give temporary relief especially in generalised cases The dose is 0 1 mg /kg body weight injected by a saline drip intravenously on alternate days for 3 to 6 injections There are often general toxic reactions such as rigors or vomiting within a few hours of the injection A second course must not be given at less than 8 weeks interval The blood must be examined to exclude leucopenia or thrombocytopenia before a second course is given

## Splenic Anæmia

### (*Banti s Disease*)

**Definition** A disease characterised by anæmia leucopenia enlargement of the spleen cirrhosis of the liver and gastro intestinal hæmorrhages

**Etiology** The cause is unknown Some authorities differentiate splenic anæmia from Banti s disease maintaining that cirrhosis of the liver does not occur in the former others deny the concept that splenic anæmia or Banti s disease is a primary disease of the spleen They believe that the condition is one of congestive splenomegaly and that the pathological findings are always due to mechanical obstruction of blood flow in the portal system the commonest cause being hepatic cirrhosis and in other cases there is extrahepatic portal obstruction due to thrombosis trauma or a pancreatic cyst

**Pathology** The spleen is enlarged and smooth It is firm on section fibrotic and the sinuses are much dilated Gandy Gamma nodules result from periarterial hæmorrhages in which fibrotic nodules form containing crystalline or amorphous iron pigments The liver may be slightly enlarged or small and cirrhotic The splenic veins are seen to be tortuous and dilated at splenectomy Phlebitis is seen in some cases in the splenic and portal veins and the œsophageal and gastric tributaries are dilated

**Clinical Findings** The patient is usually a young adult male The disease is rare in children and after middle age The onset is insidious The first symptoms of ill health may be lassitude pallor epistaxis hæmatemesis melæna or enlargement of the abdomen Banti s disease is described as passing through three stages —*The first stage* There is anæmia and enlargement of the spleen but no jaundice The spleen may weigh as much as 25 oz and yet not be palpable The blood There is a hypochromic anæmia with a low colour index The white cells show a leucopenia with a relative lymphocytosis The fragility of the red cells is normal The blood platelets may be increased or diminished constituting a thrombocythæmic or a thrombocytopenic variety of the disease *The second stage* After 2 or 3 years the liver

enlarges and there is slight jaundice *The third stage* In another 2 or 3 years the liver shrinks and ascites appears It must be understood that all cases do not pass through these stages *Severe hæmorrhage* may occur from the œsophagus, stomach intestines kidneys or nose and purpura may be seen The lymphatic glands do not enlarge

**Differential Diagnosis** In establishing the diagnosis it is necessary to consider other causes of severe anæmia of epistaxis and internal hæmorrhages of tumours in the left hypochondrium and of enlargement of the liver and spleen or of ascites The blood count demonstrates the anæmia The tumour in the left hypochondrium presents the characteristic features of an enlarged spleen A gastric ulcer and carcinoma of the stomach or colon may present difficulties but an opaque meal usually will exclude these lesions In acholuric jaundice the fragility of the red cells is increased and bleeding from mucous surfaces does not usually occur The supporters of the view that Banti s disease is a separate entity state that in cirrhosis of the liver the spleen is not usually enlarged before the liver enlarges and the splenic enlargement in cirrhosis is not usually as marked as in Banti s disease With primary thrombosis of the portal or splenic vein there is usually leucocytosis and the abdominal symptoms are more acute

**Course and Complications** The course is slowly progressive as described above although sudden death may occur from hæmorrhage Intercurrent infections may ensue

**Prognosis** This is very grave if untreated the disease is almost always fatal

**Treatment** Iron should be given in adequate doses such as ferri et ammon cit gr 30 t i d for eight weeks This may be combined with X ray treatment to the spleen In cases of hæmorrhage a drip transfusion may save the patient s life although usually only temporarily as recurrences are very liable to occur at intervals of months or years *Various types of portacaval anastomotic operations* may be successful in lowering portal hypertension and preventing recurrences of hæmorrhage but they are often technically impossible Splenectomy is of doubtful value and the operation is not devoid of risk The occurrence of post operative thrombosis in cases with a high platelet count has been reduced by the use of anticoagulants Further severe hæmorrhages are liable to occur after splenectomy in at least 50% of patients who suffered from hæmorrhage before the operation

## THE LIPOIDOSES

Under this heading are classified three diseases characterised by a disturbance of lipoid metabolism

### Gaucher's Disease

*(Cerebroside Lipoidosis)*

**Definition** A rare disease characterised by enlargement of the spleen, the presence of Gaucher cells in the spleen and other organs and a tendency to a familial incidence

**Etiology** The cause is unknown It is regarded as an inborn error of lipoid metabolism The tendency to the disease is thought to be transmitted as a simple dominant trait It may remain sub clinical the individual then acting as a carrier although able to transmit the clinical disease In succeeding generations it is transmitted at an earlier age until the infantile type is reached with lesions in the central nervous system which is rapidly fatal

**Pathology** The spleen is enlarged and the capsule thickened Infarcts may be seen on section Microscopical examination reveals the typical Gaucher cells They are enlarged reticulum cells derived from the reticulo endothelial system containing a lipoid kerasin Similar cells may be found in the liver mesenteric glands and bone marrow and in the infantile type in the brain and the lungs

**Clinical Findings** The onset is in infancy or in childhood or later and females of the Jewish race are especially affected The patient may complain of abdominal swelling or discomfort of pains in the muscles and bones and of a tendency to bleeding from the nose or gums

*On Examination* The enlarged spleen is usually found on routine examination The liver may be just palpable or considerably enlarged There is often pigmentation of exposed areas of the skin and symmetrical pigmentation of the legs in stripes there may be slight jaundice A brown yellow fatty thickening (pinguecula) may be seen on the conjunctivæ X ray examination of the bones especially of the vertebræ and femurs may show decalcification sclerosis bone absorption deformities and rarely the cystic decalcification similar to that met with in essential xanthomatosis In the early stages the lower end of the femurs may show a characteristic Erlenmeyer flask deformity due to flaring of the distal part of the femur the cortex being thinned and distended The blood There may be some degree of hypochromic anæmia and leucopenia The blood platelets are slightly diminished Later hypersplenism may develop with hæmolytic anæmia leucopenia and thrombocytopenia The blood cholesterol figures are normal Examination of the bone marrow obtained by sternal puncture shows typical Gaucher cells

**Differential Diagnosis** The diagnosis can only be established with certainty during life by splenic sternal or liver puncture and the discovery of Gaucher cells in the material removed The familial incidence age of onset enlargement of the spleen chronic course and the usual maintenance of fair health suggest the diagnosis

**Course and Complications** The course is prolonged and the patient usually survives for many years In the later stages the liver may be considerably enlarged

**Prognosis** The acute infantile variety is usually fatal within 6 months The adult type tends to be chronic Subclinical cases in which Gaucher cells are found in the bone marrow have a normal expectation of life

**Treatment** Splenectomy often relieves the symptoms and corrects the blood changes due to hypersplenism but has no effect on the course

of the disease This also applies to X ray treatment The bone pains may be relieved by X ray treatment

## Niemann-Pick Disease

*(Histiocytic Sphingomyelinosis)*

This disease affects infants usually of the Jewish race It is characterised by enlargement of the spleen, liver and superficial lymph glands The face has a mongoloid expression Black spots may be seen on the tongue The Niemann Pick foam cells are present in various organs of the body including the central nervous system There is an accumulation and retention of diaminophosphatide and sphingomyelin The skin is pigmented on exposed areas and the blood shows a hypochromic anæmia with lipoid vacuoles in the white cells The blood cholesterol is slightly raised in a few cases The blood serum does not show an increase of sphingomyelin Cells obtained by sternal or splenic puncture show a typical foam appearance The disease appears to be related to the Tay Sachs syndrome It is usually fatal before the age of two years and no cure is known

## Hand Schuller-Christian Disease

*(Xanthomatosis of Bones)*

This is another disorder of lipoid metabolism (cholesterol esters) which usually affects males Accumulations of lipoid material with formation of granulation tissue or fibrotic areas containing cholesterol crystals occur in bones, especially in the membrane bones of the skull Xanthoma nodules also occur in long bones which have a cyst like appearance in X ray films The spleen liver kidneys and lymph glands are often enlarged The total blood cholesterol is normal or a high normal The disease shows itself in early childhood and is characterised by bony softening exophthalmos and diabetes insipidus Granulation tissue involving the orbit results in exophthalmos and when it invades the sella turcica diabetes insipidus results Small xanthomatous nodules may be seen on radiological examination of the lungs resembling miliary tuberculosis Rarely the disease first shows itself in adult life the bony changes being chiefly in the long bones and exophthalmos is not necessarily present

## Enlargement of the Spleen

The causes of enlargement of the spleen may be classified as follows 1 *Protozoal infections and parasitic worms* such as malaria kala azar and schistosomiasis 2 *Bacterial infections* such as enterica group organisms streptococci in septicæmia etc 3 *Hæmopoietic and reticulo endothelial diseases* such as pernicious anæmia leukæmia Hodgkin s disease Gaucher s splenomegaly acholuric jaundice polycythæmia rubra splenic neutropenia etc 4 *Specific infective granulomata* as in tuberculosis and syphilis 5 *Deficiency diseases* such as rickets 6 *Vascular disturbances* such as infarction passive

hyperæmia from torsion of the pedicle thrombosis of the splenic vein cirrhosis hepatis pressure of enlarged glands on the portal vein and very rarely congestion due to heart failure 7 *Cysts* These may be classified as parasitic i e a hydatid cyst and non parasitic The latter are sub divided into true and false cysts True cysts may be dermoid hæm angioma lymphangioma endothelioma (serous) or due to polycystic disease False cysts may result from trauma hæmorrhage malaria infarcts or degeneration 8 *Tumours* such as primary or secondary sarcoma or secondary carcinoma The spleen may be invaded by a direct extension of a carcinoma of the stomach or pancreas Simple tumours are rare such as a fibroma lymphangioma and hæmangioma 9 *Abscess* such as a septic infarct 10 *Degeneration* as in amyloid disease

The signs of enlargement of the spleen are as follows A tumour may be seen or felt in the left hypochondrium The spleen enlarges downwards and forwards towards the umbilicus and may extend also downwards towards the anterior superior spine of the left ilium and to the right of the mid line A notch can usually be felt on its anterior border The loin is not filled by the tumour It moves slightly with respiration unless fixed by adhesions It is dull on percussion and the dulness extends backwards to fuse with the normal area of splenic dulness over the left lower che t behind There is no band of intestinal resonance running across the dull area but the flank behind the tumour is usually resonant

Differential Diagnosis Other swellings in the left hypochondrium which must be excluded are a renal or suprarenal tumour a growth of the stomach or colon a pancreatic or ovarian tumour and tuberculous peritonitis With renal tumours there are generally urinary changes and the characteristic signs are present An opaque meal or enema will assist in excluding a growth in the stomach or colon Pancreatic tumours when palpable are usually cysts appearing on the surface near the midline An ovarian tumour could only be mistaken for a very large splenic tumour but pelvic examination will indicate its origin Tuberculous glands with peritonitis do not usually give the typical signs of an enlarged spleen

## Rupture of the Spleen

This is a rare event It may follow a blow over the spleen or a crushing accident The spleen may rupture spontaneously as a com plication of malaria infarction typhoid fever pregnancy or nitro benzene poisoning Clinically the patient suffers from severe pain and shock with the signs of internal hæmorrhage and a laparotomy is nece sary to save life

## Perisplenitis

This occurs especially in connection with splenic infarction The patient complains of pain over the spleen increased on respiration A coarse rub may be heard over the spleen at the back of the chest or in the left hypochondrium

## Erythrocytosis

(*Secondary Polycythæmia*)

**Definition** An increase in the number of red cells in the blood due to some known stimulus

**Etiology** The bone marrow is provoked to increase its output of red cells under various conditions interfering with the oxygen supply such as *high altitudes, congenital* heart disease pulmonary arterio sclerosis (Ayerza s disease) pulmonary arterio venous aneurysm emphysema heart failure Cushing's syndrome, chemical substances such as cobalt etc

## Erythræmia

(*Polycythæmia Rubra or Vera Vaquez's Disease Osler's Disease*)

***Definition*** *A condition characterised by an increase in the number of red cells in the blood, with splenomegaly*

**Etiology** The cause is unknown It has been suggested that there is thickening of the vessels supplying the bone marrow, the resultant anoxia stimulating erythropoiesis

**Pathology** There is hyperplasia of marrow in the long bones The spleen is enlarged and may contain infarcts

***Clinical Findings*** *The patient is usually middle aged, of either* sex, other members of the same family may be similarly affected The onset is insidious with headache lassitude, giddiness, dyspnœa and insomnia Epistaxis or a hæmorrhage from the stomach uterus and rarely from the lungs may occur The patient may notice that his face and hands are becoming high coloured There may be severe pain in the bones and abdominal pain due to mesenteric thrombosis

*On Examination* The face is typically brick red and the ears lips hands and fingers may be cyanosed but are not clubbed The spleen is enlarged to a variable extent The heart is not usually enlarged The blood The total volume is two to three times the normal Red cells *7 to 12 millions per c mm* Hb 125 to 150% C I 0 8 to 0 9 There is polychromasia and a few normoblasts and reticulocytes are present The platelets may be increased White cells 15 000 to 20 000 per c mm Polymorphonuclears 70 to 90% A few myelocytes are present The sedimentation rate is delayed owing to the increased packed cell volume *The urine There is often a trace of albumin and a few casts* may be present Acholuric jaundice may be present The blood pressure may be raised as in polycythæmia hypertonica of Gaisbock (see p 280) In these cases there is usually no splenomegaly

**Differential Diagnosis** Erythræmia must be distinguished from erythrocytosis for which some causative *factor can be found* Leuco cytosis and increased platelets favour the diagnosis of erythræmia Enterogenous cyanosis is recognised by spectroscopic examination

**Course and Complications** The disease pursues a chronic course which is interrupted by remissions In some cases the red cell count may drop to 3 millions with an increase of *immature* white

cells resulting in a myeloid leukæmia or an aplastic anæmia before death occurs Complications include cerebral thrombosis and heart failure Cirrhosis of the liver may occur constituting the Moss syndrome

**Prognosis** Death usually takes place 8 to 8 years after the diagnosis has been made but the radioactive phosphorus treatment has improved the prognosis

**Treatment** The patient should not eat any meat liver kidneys meat soups or eggs in order to reduce the iron content of the diet Vene section affords temporary improvement 500 ml should be removed twice a week until the Hb falls to about 80% red cells to about 5 millions and the hæmatocrit reading to about 45% Phenylhydrazine is seldom used now Splenectomy is not advisable Some improvement has been obtained after X ray treatment of the long bones if care is taken to avoid stimulation of the bone marrow or the production of aplastic anæmia Favourable results have been obtained by administration of radioactive phosphorus The initial intravenous dose is usually 3 8 millicuries If the red cell count remains above 6 000 000 per c mm a second injection may be given 6 months later The initial dose by mouth of $P^3$ is 5 to 8 millicuries the red cells do not fall for 3 to 6 weeks and the total effect is not produced for 3 months If a second dose is required 3 to 5 millicuries are given The $P^3$ should be given in the morning after a twelve hour fast and no food should be taken for 3 hours subsequently Potassium salts of $P^{32}$ must not be given by mouth and for 2 days before and after taking the dose very little milk or potassium containing foods should be taken There is a latent period of about 100 days before the effect on the red cells is noted The metal is concentrated in the bone marrow where it constantly bombards the hæmopoietic tissue Alternatively nitrogen mustard may be given after a preliminary venesection The dose is mg 10 to 20 intra venously every 4 weeks until an adequate fall in the red cell count is obtained Nausea and vomiting may be troublesome side effects of the drug T E M (triethylene melamine) can be given by mouth as enteric coated tablets the dose being mg 5 a week The side effects are less troublesome than with nitrogen mustard It requires strict hæmato logical control

## Enterogenous Cyanosis

*(Sulphæmoglobinæmia Methæmoglobinæmia)*

**Definition** Cyanosis due to the presence of sulphæmoglobin or methæmoglobin in the blood

**Etiology** Stockvis in 1902 described what he called enterogenous cyanosis in a patient suffering from chronic dysentery whose blood contained methæmoglobin In these cases it is thought that nitrites are absorbed from the intestines into the blood and these reduce the hæmoglobin The reduced hæmoglobin combines with the small amount of sulphuretted hydrogen normally present and sulph Hb or met Hb is formed A nitrifying organism (nitroso bacillus) has been isolated from the saliva and fæces in some cases and reducing substances

are present in the serum, urine and saliva of patients suffering from the disease In 1925 Snapper suggested that the hæmoglobin is sensitised by aniline drugs and then unites with sulphides absorbed from the intestines Potassium chlorate, Bromo seltzer acetanilide (antifebrin), antipyrin (phenazone) phenacetin, nitrites and sulphonal may cause methæmoglobinæmia Nitrates in drinking water used in making up artificial feeds may cause the syndrome known as "blue water babies" Similar cases have resulted from absorption through the skin of aniline *dye from marking ink on infants napkins* *Sulphæmoglobinæmia* may occur in connection with sulphonamide therapy Whether sulphæmoglobinæmia or methæmoglobinæmia develops in sensitised patients appears to depend upon the degree of constipation present, but this is not always so A few cases of familial idiopathic methæmoglobinæmia have been described differing from enterogenous cases by a normal nitrite content in the blood urine and saliva whereas in enterogenous cyanosis the nitrite content may be increased

**Clinical Findings** The patient is usually an adult of either sex The onset of symptoms is insidious with headache weakness fainting attacks and nervous instability Nausea vomiting and abdominal pains may be complained of There is mild or transitory constipation with methæmoglobinæmia and severe and chronic constipation with *sulphæmoglobinæmia*

*On Examination* The patient has a peculiar cyanosis which is mauve lavender in colour in sulphæmoglobinæmia and blue chocolate brown in methæmoglobinæmia This again is not always so No physical cause for the cyanosis can be found on clinical examination The blood Spectroscopic examination shows the band of met Hb or sulph Hb between the C and D lines of the spectrum further spectroscopic tests differentiating them The pigment absorption band may be seen with a hand spectroscope through the lobe of the ear but for accurate determinations laked diluted blood should be examined in the laboratory and special differentiating tests applied *The pigment is not present* in the serum or in the urine A clinical test to distinguish between the types is as follows If the brown muddy colour disappears from the blood on standing exposed to the air for several hours the condition is methæmoglobinæmia due to an ingested chemical If the colour remains it is due either to sulphæmoglobinæmia or to congenital methæmoglobinæmia

**Differential Diagnosis** Other causes of cyanosis especially *diseases* of the heart and lungs are excluded on clinical examination

**Course and Complications** The course in the enterogenous cases is prolonged and complete *recovery is doubtful* In some cases intermissions and relapses correspond with periods of freedom from, or affection with marked constipation

**Prognosis** The condition is not fatal but a permanent cure is unusual

**Treatment** In all cases an exhaustive search should be made to exclude the use of such sensitising drugs as phenacetin antifebrin (acetanilide) sulphonal and potassium chlorate *The constipation*

must be treated by the regular use of laxatives avoiding those containing sulphur The cyanosis of methæmoglobinæmia can often be abolished by the administration of a methylene blue pill gr 2 3 or 4 times a day A maintenance dose of gr 1 every other day may prove sufficient In the congenital variety of methæmoglobinæmia with an intake of mg 800 ascorbic acid daily for a month the percentage of methæmoglobin in the blood can usually be sufficiently lowered to abolish cyanosis but on discontinuing the ascorbic acid the cyanosis reappears The change from methæmoglobin to hæmoglobin can also be effected in vitro

## PURPURA

**Definition** A condition characterised by extravasation of blood into the skin and mucous membranes due to capillary hæmorrhage

**Etiology** Purpura is a symptom occurring in many pathological states For clinical purposes it is divided into a primary and secondary group Primary or idiopathic purpura is also known as *the hæmorrhagic diathesis* it arises apart from any known cause Secondary or symptomatic purpura is a symptom of some recognisable disease The following varieties are thus described

1 *Primary or Idiopathic Purpura* The cases fall into two groups (*a*) With a low platelet count Purpura hæmorrhagica (*b*) With a normal or slightly reduced platelet count but with an abnormal capillary endothelium Purpura simplex Purpura rheumatica Henoch s purpura Allergic purpura These are all classified as anaphylactoid purpuras Purpura fulminans is also non thrombo cytopenic

2 *Secondary or Symptomatic Purpura* This may be due to (*a*) Bone marrow defects with a low platelet count Blood diseases include pernicious anæmia aplastic anæmia and leukæmia Secondary carcinomatosis of bone Chemical causes such as iodides mercury quinine belladonna ergot chloral hydrate gold arsenic sulphonamides turpentine Tridione phenacetin Sedormid salicylic acid sodium para aminosalicylic acid benzol and snake venom Exposure to X rays radium and atomic bomb explosion (*b*) Alterations in the capillary endothelium Infections such as septicæmia and infective endocarditis Fevers such as cerebro spinal fever typhus fever measles small pox or scarlet fever Cachexia as in old age chronic nephritis tuberculosis and carcinoma Mechanical causes such as pressure on the skin whooping cough violent vomiting or epilepsy Nutritional causes such as lack of vitamin C or P (*c*) Deficiency of prothrombin as in jaundice cirrhosis hepatis or dicoumarol poisoning

**Pathology** In hæmorrhagic purpura all the elements of the blood pass through the capillaries In anaphylactoid purpura the serum alone may pass out into the tissues in some parts of the body whereas the lesions in other sites are definitely purpuric In purpura simplex there may be extravasation of red cells only The lesions produced are thus variable pinpoint spots constitute petechiæ the purpuric spots are about 1 to 3 mm in diameter and larger extravasations

several inches across are known as ecchymoses The bleeding may occur into or under the skin, into or from mucous or serous membranes into the interstitial tissue of internal organs and into the eye It must be clearly recognised that purpura does not depend merely upon deficiency of platelets in the blood, *as there may be a complete absence* of platelets without hæmorrhages The platelets are much diminished in purpura hæmorrhagica ( essential " thrombopenia) and to a lesser degree in the other varieties of purpura, depending upon the severity of the bleeding The platelets are produced in bone marrow from megakaryocytes and destroyed by the spleen They probably protect weak surfaces in the capillaries by forming a layer over the intima at such spots, and thus help to prevent extravasation of blood The essential lesion in purpura appears to be a temporary alteration in the permeability of the capillary endothelium This may be due to toxins or to proteins to which the patient is sensitive but this is uncertain As long as the capillary endothelium is normal extravasation of blood does not occur, even with very low platelet counts The coagulation time of the blood is normal in purpura although the clot retracts slowly the bleeding time is much prolonged in primary purpuras when the platelet count is low After splenectomy the platelet count rises very rapidly, due partly to an increased output from the marrow and partly to lack of destruction by the spleen, but it may return to its original low level although the patient is relieved of his symptoms

## Purpura Hæmorrhagica

(*Morbus Maculosus of Werlhof Essential Thrombopenia Purpura Thrombopenia Thrombocytolytic Purpura Primary Hypersplenism Thrombocytopenic Purpura* )

**Definition** A disease characterised by hæmorrhages into the skin mucous membranes and internal organs with a low platelet count

**Etiology** Two types are described 1 Idiopathic 2 Secondary to drugs such as Sedormid iodides gold arsenic quinine etc , *and to* infections especially pneumococcal and meningococcal

**Pathogenesis** There appear to be two varieties of the idiopathic type In one which is more common the spleen is primarily at fault the platelets being sequestrated in the organ to an abnormal degree—hypersequestration—or there is also increased destruction of platelets by clasmocytes The bone marrow shows an increased number of *megakaryocytes In the second variety there is* a thrombocytopenic factor in the plasma The secondary variety due to drugs and infection results from bone marrow damage reducing the production of platelets In Sedormid sensitivity an antibody is found which causes agglutination and lysis of platelets

**Clinical Findings** The disease may show itself in an acute or chronic form The patient is often about the age of puberty but the purpura may occur at any age and more frequently in women The onset is often sudden with a hæmorrhage from the nose *stomach*,

kidneys or uterus Careful enquiry must be made to exclude drug sensitivity especially that due to Sedormid

*On Examination* Purpuric spots and ecchymoses may be found on the body the gums may bleed but are not spongy as in scurvy The spleen is enlarged in about one third of the cases The temperature is often raised The blood The platelets are usually much reduced and may disappear altogether although the initial symptoms may appear with a platelet count of 100 000 per c mm The coagulation time and prothrombin time are normal The bleeding time may be prolonged to several hours The retraction of clot in shed blood is poor There is usually a hypochromic anæmia and the reticulocytes may be increased up to 10% or more A few nucleated red cells may be seen There may be a leucocytosis or a leucopenia with relative lymphocytosis Sternal puncture differentiates between the idiopathic type due to hypersplenism and the secondary type resulting from hypoplasia of bone marrow It also excludes an acute leukæmic infiltration of the bone marrow

**Differential Diagnosis** Hæmophilia can usually be excluded on clinical grounds The platelet count and bleeding time are normal and the coagulation time is delayed Hereditary purpura hæmorrhagica (constitutional hæmogenia) is a disease resembling purpura hæmor rhagica clinically but differing in its tendency to hereditary trans mission its preference for the female sex and the often fatal results of splenectomy In only about 50% of cases is the platelet count low In scurvy the hæmorrhages are usually in deeper tissues such as the thighs the gums are spongy and there is a history of avitaminosis In acute leukæmia the clinical picture may closely resemble that of the hæmor rhagic diathesis The blood count usually serves to differentiate but there may be a leucopenia with relative lymphocytosis in acute leukæmia The capillary resistance test of Hess may be of value in a doubtful case A circle is marked out on the forearm an inch in diameter with its upper margin 2 inches below the bend of the elbow The upper arm is com pressed with the armlet of a sphygmomanometer the pressure being maintained for 15 minutes mid way between the systolic and diastolic readings The cuff is then removed and 5 minutes later the number of hæmorrhagic spots is counted which have appeared within the circle Anything above 20 is abnormal and indicates increased capillary fragility Some cases diagnosed as essential thrombopenia prove to be congenital nasal telangiectases

**Course and Complications** The course may be rapidly fatal or more prolonged with intermissions or definitely chronic with recurrent hæmorrhages and purpura for several years In many cases death is due to subdural hæmorrhage Cerebral or cerebro spinal hæmorrhage may occur as a complication

**Prognosis** This is always very grave in acute cases Chronic cases are more benign and there is a tendency to spontaneous improvement

*Treatment* Splenectomy is indicated when it has been demonstrated that the spleen is primarily at fault This must be preceded by a series of blood transfusions Relapses occasionally occur these are either due

to accessory splenic tissue in the body at some other site or to a thrombocytopenic plasma factor being present Cortisone in doses of mg 200 to 300 daily may produce remissions lasting for several months and is worth a trial before splenectomy is advised In secondary cases also treatment should be directed to the primary cause If due to *gold or arsenic* British Anti Lewisite (B A L ) should be given It is put up as a 5% solution of dimercaprol in arachnis oil containing 1% v/v of benzyl benzoate Each 2 ml ampoule contains mg 100 B A L The dose for an adult is mg 100 intramuscularly four times during the first 24 hours at four hourly intervals On the second to fourth days mg 100 morning and evening, and on the fifth and sixth days mg 100 daily

## Purpura Simplex

**Clinical Findings** This disease was very common 20 or 30 years ago but is now rarely seen It is considered to be an allergic manifestation The patient is usually a child or young adult who gives a history of slight malaise with perhaps headaches fleeting pains in the joints *or diarrhœa The patient complains of lassitude with pains in* the shoulders and legs

*On Examination* Small purpuric spots are seen chiefly on the extensor surface of the legs The trunk arms and rarely the face may be involved The spots come out in crops and the temperature *may be slightly raised Hæmorrhages do not occur from the* mucous membranes The blood The platelets are usually normal The coagulation time is normal The bleeding time is normal

**Course and Complications** 5 or 6 weeks may elapse before the spots finally disappear Recurrences are often noted

**Prognosis** *This is good*

**Treatment** The patient should be in bed until the purpura disappears Treatment is symptomatic

## Schonlein's Purpura

(*Anaphylactoid Purpura Allergic Purpura Purpura Rheumatica Peliosis Rheumatica*)

**Etiology** The cause is unknown There is no evidence that Schonlein s purpura is a rheumatic infection It may be due to sensitivity to proteins or to a streptococcal *infection*

**Pathology** At some sites in the body the capillaries allow transudation of plasma and at others diapedesis of red cells occurs

**Clinical Findings** The patient is often a young adult male There may be fever and sore throat at the onset with pain in various muscles and joints *Swelling of joints may also be noted*

*On Examination* The temperature may be raised to about 100 F for the first few days of the illness The affected joints such as the knees or ankles are slightly swollen and tender but the *skin is* not discoloured The skin The cutaneous changes include purpuric spots *usually on the extensor surfaces of the* legs and urticarial wheals Angio neurotic swellings may appear in the legs feet face or hands

The blood Usually the platelets are not diminished and the bleeding and coagulation times are normal

**Differential Diagnosis** The fever sore throat and joint pains are suggestive of acute rheumatism and the hæmaturia of acute nephritis The purpuric spots and urticarial swellings are diagnostic and there is no response to salicylates

**Course and Complications** The course may be prolonged for several weeks and relapses occur In some cases there are intestinal symptoms characteristic of Henoch s purpura The urine may contain albumin and blood

**Treatment** The patient must be kept in bed Antihistamine drugs such as Benadryl mg 50 capsule t i d or Thephorin mg 25 tab t i d may be given Salicylates do not relieve the joint pains In cases associated with nutritional deficiency vitamin P should be administered in the form of Hesperidin tab mg 150 four times a day Later any septic focus in the mouth or elsewhere should receive treatment

## Henoch s Purpura

**Definition** This is a more severe form of Schonlein s purpura characterised by abdominal crises due to an exudation of plasma or blood into the wall of the intestines

**Clinical Findings** The patient may be an infant or adult who is seized with severe abdominal pain vomiting diarrhœa or constipation and at times pains in the joints

*On Examination* The abdomen is rigid during the attack There may be no purpura or careful search may reveal the presence of a few spots Urticaria and swelling of the joints may also be present The blood The platelets are usually normal and there is no change in the bleeding or coagulation times Blood and mucus may be passed per rectum and the temperature is raised

**Differential Diagnosis** The disease is liable to be mistaken for an intussusception in an infant or for a mesenteric thrombosis or other variety of abdominal emergency in an adult A very careful search should be made for purpuric spots before any operation is performed

**Course and Complications** An attack usually passes off in a day or so Complications include hæmaturia intussusception and cerebral hæmorrhage Relapses are prone to occur

**Prognosis** This is always serious

**Treatment** The patient must be kept in bed and an icebag applied locally to the abdomen to relieve pain If this is not successful tnc chlorof et morphin co (B P C ) m 5 to 10 may be given to an adult and repeated if necessary Antihistamine drugs and vitamin P are worthy of trial administered as above

## Purpura Fulminans

A hyperacute variety of purpura which causes death in 1 to 5 days It has been described following scarlet fever Infants are chiefly affected the temperature is raised and large subcutaneous ecchymoses are seen The blood shows an anæmia with a normal number of platelets The

Waterhouse Friderichsen syndrome due to meningococcal septicæmia is a closely allied condition

### The Secondary Purpuras

The conditions with which purpura may be associated have been enumerated on p 543

### Hæmophilia

**Definition** A familial disease characterised by a tendency to bleed severely from trivial injuries

**Etiology** Hæmophilia is due to a "failure or delay in blood thromboplastin formation caused by absence or deficiency in the blood of antihæmophilic globulin" It occurs in males but is transmitted by females as a Mendelian sex linked recessive trait (Nasse s law) When a hæmophiliac man and a normal woman marry the male offspring will be normal but the females will carry the hæmophilic trait If such a female carrier marries a normal man, the chances are about equal whether their sons are hæmophiliacs or their daughters carriers of the trait If however a hæmophiliac marries a female carrier of the trait the daughters may be true hæmophiliacs

**Clinical Findings** The history frequently suggests the presence of 'bleeders' in other male members of the family, and they can often be traced back for more than one generation The peculiarity is often noted during the second year of life, a trivial injury causing excessive bruising or bleeding if the skin is abraded or cut Extraction of a tooth may give rise to serious bleeding

*On Examination* Ecchymoses may be seen under the skin or a hæmatoma felt A joint may be distended with blood especially the knee or elbow The affected joint is painful and the skin over it red The temperature then is usually raised to about 100 F Muscular hæmorrhages may cause pain by nerve pressure Vitreous, retinal and orbital hæmorrhages have occasionally been recorded The blood There may be a hæmorrhagic anæmia The platelets are not deficient The bleeding time is normal or slightly prolonged a prick not causing prolonged bleeding as does a scratch as the minute wound of the prick is sealed by a mass of platelets It is therefore quite safe to perform a vein puncture The coagulation time is prolonged to 5 to 8 times the normal but it may revert to nearly normal between the attacks The blood of a normal person contains antihæmophilic globulin and when added to the blood of a hæmophilic patient shortens its clotting time This is used as a test the blood of a suspected hæmophiliac is added to that of a known hæmophiliac and the shortening of the clotting time is compared with that obtained with normal blood During the active stages of the disease the bone marrow may show an increase in megakaryocytes and megakaryoblasts

**Differential Diagnosis** Hæmophilia must be distinguished from simple obstinate bleeding such as may occur after a dental extraction This may affect either sex and there is no history of hæmophilia In purpura hæmorrhagica the prolonged bleeding time and thrombopenia are characteristic In *pseudohæmophilia* there is a prolonged bleeding

time but no other abnormality in the blood Severe bleeding is liable to occur from wounds from the nose from the gastro intestinal and female genital tracts *Thrombasthenia* is characterised by a tendency to purpura and hæmorrhages The bleeding time and platelet count are normal but clot retraction is defective *Fibropenia* is a rare disease in which the blood contains no fibrinogen and so will not clot In the hæmorrhagic diseases of the newborn the bleeding occurs during the first few days of life usually with melæna

**Course and Complications** There is a tendency for the condition to improve with the passage of years Joints which have been affected with a hæmorrhage may become ankylosed by fibrous tissue

**Prognosis** This is always very grave many hæmophiliacs failing to survive to adult age

**Treatment** The patient must be protected from all varieties of external trauma A small local hæmorrhage may be sometimes arrested by the application of liq adrenalin hydrochlor on cotton wool or by normal human blood applied on wool after removal of the clots from the wound If this fails a sterile 1 in 10 000 solution of Russell s viper venom (Stypven or Rusven) should be applied on gauze Major surgery should be avoided in all cases of hæmophilia unless it is absolutely essential owing to the high mortality rate In all operations including dental extractions a plasma transfusion of 1 to 2 litres of fresh plasma obtained from a suitable donor should be given immediately before hand A second transfusion may be required after a minor operation After a more severe operation daily transfusions of 500 to 1 500 ml may be required for 2 weeks as the wound is slow to heal Alternatively fresh frozen plasma stored at − 20 C may be used Such plasma keeps when frozen solid for at least a month A fibrinogen fraction containing antihæmophilic globulin is obtainable from the Lister Institute London which is stable at room temperature for several months It contains about 5 times as much antihæmophilic globulin as does normal plasma The average dose is 200 ml The Cohn plasma fraction 1 contains fibrinogen and antihæmophilic globulin but is said not to be so reliable Hæmarthrosis is treated by elevating the joint and applying bandages wrung out in iced water In some cases it is necessary to aspirate the joint Tooth sockets should not be plugged or sutured

### Christmas Disease

This disease named after a patient who suffered from it resembles hæmophilia in clinical and laboratory findings In 1952 it was shown that in certain patients suffering from hæmophilia antihæmophilic globulin is present in the plasma The prolonged clotting is due in these cases to a lack of another factor necessary to the formation of thromboplastin This so called Christmas factor is stable so that transfusions with stored blood are effective in its treatment

### Hæmorrhagic Thrombocythæmia

This is a rare condition characterised by a great increase of mega karyocytes and platelets in the bone marrow and by a blood platelet

count of 3 millions or more Thrombosis of the larger veins of the legs and of the mesenteric vessels is liable to occur, also subcutaneous hæmatomata Treatment with radioactive phosphorus has been effective in some cases

## Hæmorrhagic Disease of the Newborn

### (*Melæna Neonatorum*)

**Definition** Spontaneous hæmorrhages in newborn infants They are often premature

**Etiology** The cause is hypoprothrombinæmia due to vitamin K deficiency developing a few days after birth and ceasing usually after the eighth day

**Clinical Findings** During the first 4 to 10 days of life hæmorrhages occur from the umbilicus, stomach or the intestines There may also be ecchymoses or bleeding from the urinary tract or vagina The infant appears fretful does not take its feeds well and may be jaundiced The blood The platelets are not diminished The coagulation time and bleeding time are prolonged There is hypoprothrombinæmia

**Differential Diagnosis** Hæmophilia does not often manifest itself at such an early age Localised hæmorrhages may be due to trauma such as a cephalhæmatoma In erythroblastosis fœtalis Winckel s disease and Buhl s disease jaundice is seen Syphilis and umbilical sepsis may give rise to similar symptoms

**Prognosis** The outlook is grave if the patient fails to respond to adequate treatment

**Treatment** *Prophylactic* The mother is given tab acetomenaphthone mg 5 5 tabs 12 to 4 hours before the baby is born and the baby is given mg 5 by mouth on the first day of its life

*Curative* The baby is given an intramuscular or intravenous injection of Kayvisyn mg 1 in 1 ml every 6 to 12 hours until the bleeding stops In severe cases a blood transfusion of 60 ml of Rhesus negative blood should be injected intravenously and repeated the next day The injection may be made into the superior sagittal sinus Intramuscular injections of blood do not appear to raise the prothrombin content of the infant s blood

## Agammaglobulinæmia

Electrophoretic study of the serum proteins has shown that in some people the gamma globulin component is absent or only present in very small quantities It is not decided whether this condition is congenital or acquired The importance lies in the fact that such subjects are very liable to recurring infections These include pyoderma purulent conjunctivitis otitis media purulent sinusitis pneumonia meningitis and purulent arthritis It appears that the immune bodies are contained in the gamma globulin and the victims of this condition are unable to produce antibodies to various antigens In some cases monthly parenteral injection of gamma globulin will keep the patient free from infection

## CHAPTER VIII

## THE INFECTIOUS FEVERS

**Introductory** The diseases included in this section are those wh are admitted to isolation hospitals for treatment Cerebro spinal fe acute poliomyelitis and encephalitis lethargica are described in chapter on nervous diseases

In practice it is not necessary to send away all such cases these hospitals and in view of the risk of 'cross infection ( child being admitted with one disease and contracting anot while in hospital) it is better to nurse them at home provided t can be adequately looked after Owing to the highly specialised med and nursing attention which may be required in cerebro spinal fe scarlet fever enteric fever and diphtheria it is advisable for pati suffering from these diseases to be admitted to a special hospital

Certain terms used in describing these diseases will now be defined *Incubation period* The time between infection and the first deve ment of symptoms it is very variable and the average figures adop by the Ministry of Health and the Board of Education are gi *Prodromal period* The time between the end of the incubation pe and the appearance of the specific rash *Exanthem* The cutane eruption *Enanthem* Eruptions on mucous membranes such Koplik s spots in measles *Quarantine period* The maximum t during which a person who has been in contact with the infec may develop the disease It is usually 1 to 2 days longer than maximum incubation period *Isolation period* The time the pat must be isolated *Fomites* (fomes = tinder) Articles with whic patient suffering from an infectious fever has been in contact which convey the infection to another person

*Notifiable Diseases* The following diseases are compulsc notifiable it will be seen that other diseases than the infectious fe are included Small pox cholera diphtheria membranous cr measles whooping cough scarlet fever erysipelas typhus the ent group of fevers relapsing fever puerperal pyrexia plague cerebro sp fever acute poliomyelitis acute encephalitis lethargica acute p encephalitis tuberculosis ophthalmia neonatorum dysentery mal acute primary pneumonia and acute influenzal pneumonia

The following can be made locally notifiable in certain areas du an epidemic and for certain periods Rubella chicken pox diarrh zymotic enteritis scabies pemphigus neonatorum anthrax glan and hydrophobia

### Measles

(*Morbilli Rubeola* (*On the Continent Rubeola is synonymous wi Rubella*) )

**Definition** An acute infectious disease characterised by e catarrh and fever and followed by a typical rash

**Etiology** The cause is a virus which affects man and monkeys The disease is spread by direct contact probably through droplet infection on sneezing or coughing It is very infectious during the early catarrhal stage before the appearance of the rash *Predisposing causes* 1 Age The maximum incidence is between 8 months and 5 years 2 Absence of a previous attack Second attacks of genuine measles are very rare 3 Season Epidemics occur in the winter and spring and are more virulent and extensive in alternate years 4 Sex climate and race play no part

**Pathology** Multinucleate giant cells apparently characteristic of measles may be found in tonsillar or appendicular tissue removed by operation during the prodromal period of the disease There are no special post mortem changes Death is usually due to complications especially bronchopneumonia

**Incubation Period** 7 to 14 days, usually 10 to 11 days

**Clinical Findings** The patient is usually a child A few hours after exposure to infection he may develop symptoms of measles and even a transient morbilliform rash This is called the "illness of infection" It disappears in a day or so and the measles may then develop at the usual date The onset is with catarrh of the eyes photophobia sneezing nasal discharge and malaise A cough laryngitis or diarrhœa may also be noted A prodromal rash sometimes occurs which may be scarlatiniform or morbilliform in character

*On Examination* On the first day the patient appears flushed the conjunctivæ are injected, the temperature is raised to 99 or 100° F or over A characteristic enanthem is found Koplik s spots being seen in over 90% of cases They are small bluish white slightly raised spots (due to necrosed epithelium) on a bright red base, about the size of a pin s head seen inside the cheeks opposite the premolar teeth The areolæ are often confluent and the spots may spread over the mucous membrane of the cheeks and lips The temperature often falls to or below normal on the second or third day (remission of measles) and rises again on the evening of the day before which the rash appears (third day) to 102° F or over and continues to rise until the rash is well out In an uncomplicated case the temperature falls gradually to normal during the next 48 hours as the rash fades (see Fig 48) but the rash and fever may last for 4 or 5 days Koplik s spots usually fade with the appearance of the exanthem

The rash is first seen on the forehead near the hair and behind the ears It spreads usually within 24 hours to the face neck and trunk and to the limbs hands and feet including the palms and soles (see Fig 49) The rash at first macular rapidly forms papules of a dull red colour varying in size from about 2 to 6 mm with an irregular blotchy appearance The papules tend to coalesce and form crescentic areas whose colour fades on pressure the intervening skin being white Confluence may lead to areas of diffuse erythema especially on the back and very rarely small vesicles may form on the spots The rash begins to disappear in 2 to 5 days in the order of its appearance becoming brownish and leaving some staining which may persist for 1 to 2 weeks

This is followed by a branny desquamation in many cases The rash be slight and abortive and the skin is often moist and itching The to is usually covered with a whitish yellow fur The blood There is leucocytosis during the invasion period followed by leucopenia a relative increase in the large lymphocytes The urine Fe albuminuria is frequently found There is frequently some bronch

*Varieties* Variations from the typical case may occur T include 1 *Morbilli sine morbillis* No rash follows the typical symptoms This occurs particularly after the administration of se which has been given with a view to allowing an attenuated attac occur 2 *Hæmorrhagic or black measles* This is very rare and f Hæmorrhages occur from the mucous membranes and into the The cases so diagnosed were probably small pox Petechial hæ rhages into the spots are not of grave significance 3 *Toxic meas* The patient is overwhelmed with toxæmia characterised by delir tremors hyperpyrexia and death 4 *Suffocative measles* Ther marked dyspnœa from the onset with rales heard all over the lungs

**Differential Diagnosis** During the invasion period the disease may be mistaken for a cold influenza laryngeal diphtheria bronchitis or enteritis

The age of the patient history of exposure to infection and above all the presence of Koplik s spots are of value in making a diagnosis The rash may be mistaken for that of rubella the prodromal rash of small pox or scarlet fever syphilitic roseola a serum rash septic rash intestinal rash post vaccinal eruption drug rashes such as those due to copaiba chloral turpentine antipyrin etc or for that of typhus fever

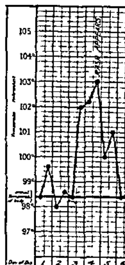


Fig 48 Temperatu Chart in Measle

**Course and Complications** The typical course has been described above but this may be seriously modified by complications The most important of these are Bronchitis bronchopneumonia laryngitis laryngeal ulceration pleu empyema diphtheria cervical adenitis blepharitis corneal ulcerat panophthalmitis herpes facialis otitis media cancrum oris and n pudendi Convulsions are not uncommon encephalitis, myel cerebellar syndromes toxic psychoses osteomyelitis and nephritis rare Nervous complications are most likely to occur between the t and fifth days of the exanthem or the eighteenth and twentieth of convalescence Enteritis ranks next to bronchopneumonia as most serious complication It may occur during the prodromal st in the eruptive period or after the rash has faded Bronchial g or pulmonary tuberculosis or general adenitis may develop as sequ Relapses are extremely rare

**Prognosis** This is very good apart from complications Measles is now a much milder disease than it was formerly The case fatality rate in Great Britain i e the number of deaths per 1,000 notifications is less than one

**Treatment** *Prophylactic* Owing to the usual mild nature of the illness prophylaxis is generally only required in weakly children under the age of 5 years An attack of measles may be prevented by the intramuscular injection within 6 days of exposure to infection of gamma globulin prepared from convalescent measles plasma and an attack may be attenuated if the gamma globulin is injected after the sixth day but before the disease appears The dose is mg 250 in 3 ml sterile water for a child under the age of 1 year Between 1 and 3 years the dose is mg 500 (6 ml ) to prevent and mg 250 to attenuate and over 3 years mg 750 (9 ml ) to prevent and mg 250 to attenuate Injection of human serum carries the risk of homologous serum jaundice Convalescent measles serum may be injected intramuscularly in doses of 5 ml for a child up to 5 years and 10 ml over 5 years In any case the passive immunity only lasts for about 3 weeks *Quarantine period* This is 3 weeks for contacts They need not be kept from school for the first week but should be isolated from all children under the age of 5 years and subsequently should be examined daily for early signs such as catarrh fever Koplik's spots and a prodromal rash *Isolation period* This is 2 weeks from the appearance of the rash

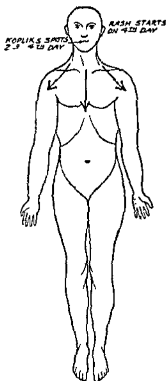



FIG 40 DIAGRAM OF RASH IN MEASLES

Rash spreads downwards back and front arms legs palms and soles for 24 to 36 hours and fades about 2 days later leaving some staining and branny desquamation The crosses indicate the usual site of the first appearance of the rash

*Curative* The patient must be put to bed immediately, and kept there for 2 to 3 days after the temperature is normal The temperature of the room should be maintained day and night at 60° to 65° F , adequate ventilation must be ensured, and if there is any bronchitis the atmosphere should be moistened with a steam kettle The bed should not face the light owing to the photophobia

If there is high fever or much irritation the skin should be tepid sponged once or twice a day If the rash does not come out well a hot sponge and hot bottles should be applied The eyes should be bathed with warm boracic lotion 2 or 3 times daily and vaseline put on the lids Gutt Albucid 10% may also be dropped into the eyes every 4 hours The inside of the mouth should be cleansed *before*

and after meals with cotton wool dipped in $H_2O_2$ (10 vols ) diluted 8 times with water The patient should be encouraged to blow his nose from time to time For otitis media penicillin should be dropped into the ear every 4 hours using 1 000 units to 1 ml distilled water Sulphadiazine should also be given by mouth in doses according to the patient s age and penicillin 300 000 units of a procaine preparation injected intramuscularly every 12 hours Convalescent serum treatment is probably of no value after the onset of the disease Encephalomyelitis is treated by lumbar puncture to relieve pressure and by the intravenous injection of 20 ml of serum from a patient convalescent from measles encephalitis In order to prevent or lower the incidence of bronchopneumonia and otitis media sulphadiazine may be given for 5 days in doses varying with the age of the patient e g 1 G t i d for a child of 5 years or penicillin may be injected intramuscularly

No rigorous disinfection of the room is necessary after measles Sheets etc should be boiled and the mattress aired for a couple of days The patient should have a bath and the hair washed before mixing with others The most important complication is bronchopneumonia for the treatment of which see p 157

## German Measles

### (*Rubella Roteln*)

**Definition** An acute infectious disease characterised by fever enlargement of glands and a typical rash

**Etiology** The cause is believed to be a virus Rubella is spread by direct contact probably by droplet infection from the naso pharynx *Predisposing causes* 1 Age Children and young adults 2 Season The greatest number of cases develop in the first half of the year 3 Absence of a previous attack Second attacks are very rare

**Pathology** There is no morbid anatomy for death practically never occurs

**Incubation Period** 5 to 21 days usually 17 to 18 days

**Clinical Findings** The patient is usually a child or young adult Often the first symptom noticed is the rash although there may be headache malaise sore throat and nasal catarrh for a day or so earlier or stiffness in the back of the neck due to the enlarged glands An acute onset with a rigor stiff neck and occipital headache may occur

*On Examination* During the invasion stage the temperature is usually raised to 99 or 100 F the eyes are somewhat pink and enlarged glands are felt at the back of the neck There are no prodromal rashes The rash appears usually on the second or third day first on the forehead and behind the ears then the face trunk and limbs are involved (see Fig 50) The rash consists of small macules and papules nearly circular and about 1 to 4 mm in diameter The colour is pink or light red The rash disappears from the face in 12 to 24 hours and there is no staining but slight desquamation may occur The rash is more confluent on the trunk and may be

almost scarlatiniform, on the extremities it is more morbilliform, irregularly blotchy in character, and frequently involves the palms and soles It usually fades completely in 86 hours The temperature falls by remissions and reaches normal on the second or third day after the appearance of the rash There may be very slight or no pyrexia (see Fig 51) The glands The following groups especially are enlarged Suboccipital posterior cervical those at the angle of the jaw, the mastoid, axillary, and inguinal glands Suppuration does not

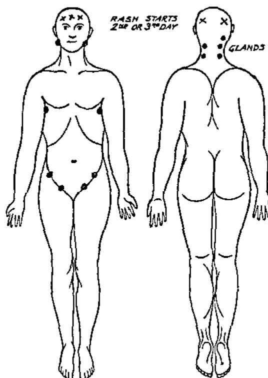



Fig 50 Diagram of Rash in German Measles

Rash spreads rapidly to face neck trunk and limbs Legs are less affected than arms The rash usually fades in 36 hours and leaves no staining The crosses indicate the usual site of the first appearance of the rash The black circles indicate glands

occur The conjunctivæ may be slightly inflamed and the spleen just palpable The blood Lymphocytosis is usually present before the rash comes out later there is leucopenia The urine is usually normal

Differential Diagnosis On the second day of the rash there may be difficulty in distinguishing the disease from scarlet fever owing to the rash having faded from the face, leaving a circum oral pallor and being confluent on the trunk The chief points indicative of rubella are The enlarged glands especially behind the neck and in the axillæ the absence of throat signs the slight degree of pyrexia the slight constitutional disturbance and later the absence of pin hole peeling Diagnosis from measles may also be difficult but the absence of severe

coryza and Koplik's spots during the invasion period of rubella and its characteristic rash and enlarged glands serve to differentiate it A morbilliform rash may occur in glandular fever, but the blood examination usually differentiates Drug enema and toxic rashes may also simulate rubella but are usually differentiated by the history and absence of typical clinical findings

**Course and Complications** The course is as described above The patient is generally quite well in a week and there are usually no complications although bronchitis stomatitis and severe muscular pains sometimes occur Encephalitis is a very rare complication In 10% to 50% of cases of rubella occurring during the first 3 months of pregnancy the child is born with a congenital defect such as cataract acyanotic heart disease deaf mutism microcephaly or mental retardation

**Prognosis** This is good

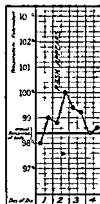


FIG 51 TEMPERATURE CHART IN GERMAN MEASLES

**Treatment** *Prophylactic* Gamma globulin prepared from convalescent German measles plasma should be injected in doses similar to those for measles (see p 554) into women contacts in the first 4 months of pregnancy *Quarantine period* for susceptible contacts is 21 days *Isolation period* The patient should be isolated for 7 days from the appearance of the rash

*Curative* The patient should be put to bed and kept there for 2 days after the rash has disappeared No special treatment is required beyond that applicable to a mild degree of pyrexia Disinfection of the patient and the room is as for measles

## Scarlet Fever

### (*Scarlatina*)

**Definition.** An acute infective disease characterised by fever sore throat a typical rash and subsequent desquamation

**Etiology** The disease is caused by group A hæmolytic streptococci Whether scarlet fever or simple streptococcal infection occurs as the result of invasion with a hæmolytic streptococcus depends upon the amount of erythrogenic toxin produced by the streptococcus and on the specific resistance of the host to that toxin The erythrogenic toxin is responsible for the rash congested fauces and red tongue Infection is spread either by direct contact probably by droplets or indirectly by fomites such as clothes toys and books by milk feeding utensils by attendants on patients and by carriers The desquamated papules are not infective *Predisposing causes* 1 Age The majority of cases occur between the ages of 5 and 10 young adults also are often affected 70% of infants according to the Dick test are susceptible between the ages of 1 and 2 but only 18% of adults after the age of 20 2 Season Epidemics have tended to occur lately in January few cases are now seen in the summer 3 Climate The disease occurs chiefly

in the temperate zones 4 Absence of a previous attack Second attacks may however, occur 5 Convalescence from diphtheria

**Pathology** There is engorgement of the fauces, dilatation of the cutaneous capillaries and cervical adenitis Inflammation may be found in the pericardium, endocardium and joints of the fingers and wrists In rapidly fatal cases the patient dies from toxæmia In other cases death may be due to complications

**Incubation Period** 1 to 8 days, usually 2 to 3 days

**Clinical Findings** At the present time scarlet fever is comparatively uncommon in Great Britain The patient is usually a young child who is suddenly taken ill with headache sore throat and vomiting

*On Examination* The child has a brightly flushed face and a pale zone around the mouth (circum oral pallor) The eyes are bright but not injected, the tongue has a thick yellowish fur, and the tonsils are red and may show some exudate A few red spots may be seen on the soft palate The temperature is raised to about 102° F and the pulse *is frequent, about 130 The rash appears on the second day, first on* the neck and upper part of the front of the chest spreading in a few hours to the trunk and limbs It is especially well marked in the axillæ groins flexures of the elbows and knees and inner side of the thighs The backs of the hands and feet and to a lesser degree the palms and soles are involved, but except for the flush, the face is not usually affected (see Fig 52) The rash consists of small bright red points on a red base (punctate erythema) the whole fading on pressure The skin feels dry and hot Definite staining may be left after the rash has faded which usually takes 4 days Small petechiæ may then be seen on the flexor aspects of the elbows and knees

Desquamation begins about the fourth day first on the face, where it is very fine and causes the powder and rouge" appearance About the seventh day it is seen on the side of the neck below the clavicles and *above the pubes It is called* pin hole *peeling* as small holes form over the sites of the red points of the rash As these enlarge and coalesce flakes are detached In the third and fourth weeks the hands and palms peel, the heels being affected last of all The tongue presents *characteristic changes on the second* day red papillæ show through the creamy fur ( white strawberry ' tongue) The tip and edges then begin to peel and on the fourth day the red strawberry' or ' ripe raspberry tongue is seen the whole tongue being bright red with *prominent papillæ* In 80% of cases a throat swab shows hæmolytic streptococci Enlarged glands are present below the angles of the jaw, and these may suppurate

The temperature This rises rapidly at the onset, falling by lysis in a mild case on the fourth or fifth day to reach normal about the eighth day or earlier (see Fig 53) The blood Leucocytosis and eosinophilia are usually present during the pyrexial stage The urine Febrile albuminuria is often noted

*Varieties* 1 *Abortive* The rash may be very slight or only seen on certain parts of the body but it is followed by desquamation and some complication may ensue

2 *Malignant or Toxic* Constitutional disturbances include prostration hyperpyrexia vomiting diarrhœa delirium insomnia and exhaustion death results in 1 or 2 days The rash is not usually well marked

3 *Septic* Severe tonsillar or palatal lesions with ulceration and necrosis are seen Otorrhœa may ensue cervical adenitis and cellulitis are present and there may be hæmorrhage from erosion of an artery

4 *Hæmorrhagic* This rarely occurs and is often fatal Hæmorrhages are seen in the skin and from the mucous membranes

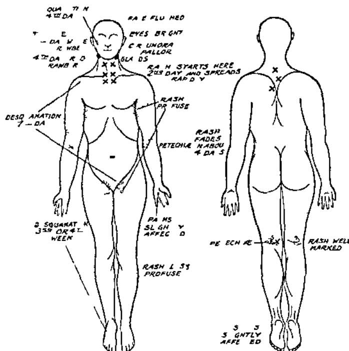



Fig 2 Diagram of Rash in Scarlet Fever
The crosses indicate the usual site of the first appearance of the rash The black circles indicate glands

5 *Surgical* This follows injuries especially burns and operations particularly on the naso pharynx The infection may enter through the throat or through the skin lesion

6 *Puerperal* This is not a special variety of scarlet fever A woman during the puerperium may contract scarlet fever but is probably not especially susceptible

Differential Diagnosis During the invasion period scarlet fever must be diagnosed from tonsillitis and diphtheria A swab should be taken from the throat if exudation is present The rapid onset of the rash usually serves to establish the diagnosis The rash must be diagnosed from the following Measles rash rubella rash septic rashes enema rash flannel rash prodromal rash of small pox erythema due

to influenza, drug rashes due to belladonna salicylates copaiba, and quinine The distinguishing characters of the rashes due to the infectious fevers are considered under their separate headings The following additional tests are of value

*The Schultz Charlton Test* or "extinction sign" One fifth of a ml of a 1 in 10 dilution of scarlatinal antistreptococcus serum is injected intradermally into the abdomen of the patient where the rash is well developed If the disease is scarlet fever, a circular area of blanching occurs in about 6 to 8 hours lasting for about 3 days No blanching results if the rash is due to other conditions A positive result is obtained in about 90% of scarlet fever cases during the first 3 days of the rash

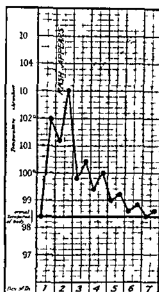


FIG 53 TEMPERATURE CHART IN SCARLET FEVER

*The Dick Test* Two tenths of a ml of the appropriate dilution of the scarlet fever streptococcal toxin are injected intradermally and the cutaneous reaction compared with that resulting from a similar injection of the control heated toxin The test is positive during the first 3 days of scarlet fever in about 90% of cases and becomes negative *in about 10 days time or later If the test* is negative at the early stage, the disease is probably not scarlet fever

**Course and Complications** The course depends on the severity of the disease and the presence of complications It is also modified by treatment with anti scarlatinal serum During the fourth or fifth week of the disease a relapse may occur, which is a repetition of the original illness The most important complications are Acute glomerulonephritis This is liable to occur about the twenty first day of the disease focal nephritis may occur in the first week Rheumatism Arthritis may appear at the end of the first week during the second week or later The fingers wrists shoulders ankles and knees are most often affected Suppuration is very rare Muscular rheumatism is not so common Other complications include otitis media meningitis cerebral abscess endocarditis pericarditis cervical adenitis persistent rhinitis bronchitis ulcerative stomatitis vaginitis and diphtheria Perforation of the *pharyngeal wall may lead to surgical emphysema* perforation of the palate may also occur Embolic or thrombotic gangrene of the extremities has also been recorded

**Prognosis** This varies with the type of disease Toxic and hæmorrhagic cases are usually rapidly fatal and septic cases frequently die The prognosis is good in simple scarlet fever although sequelæ may result from *complications such as nephritis endocarditis* or otitis media which may prove fatal several years later

**Treatment** *Prophylactic* Active immunisation by means of scarlet fever toxin is now seldom employed The prophylactic administration

to contacts of crystalline penicillin 200 000 to 500 000 units twice a day or of sulphadiazine 0 25 G t i d for 4 or 5 days is widely employed *Isolation period* The minimum isolation period for a simple case is 4 weeks but the patient must be isolated while there is any discharge from the nose ears vagina or a wound and until the nose and throat are apparently healthy Children must not return to school for 2 weeks after release from isolation A hæmolytic streptococcus is present in the throat in many cases on discharge from hospital and peeling is often not completed

*Return cases* are fresh cases of scarlet fever resulting from infection by the convalescent patient after he has been freed from isolation The infective agent in such cases is probably still present in the nose or throat A throat swab however which is negative as regards the hæmolytic streptococcus does not mean that the patient is free from infection

*Curative* Mild cases of scarlet fever can be nursed at home The patient must be kept in bed for at least 2 weeks At the onset a fluid or light diet should be given The mouth should be cleaned after each feed with some antiseptic such as glycerinum boracis on a swab A hot normal saline mouth wash will afford relief for the sore throat together with aspirin gr 5 to 10 every 4 hours Antitoxin is now seldom used except in very toxic cases Streptococcus Antitoxin—Scarlatina can then be injected intramuscularly intravenously or intraperitoneally in doses of 3 000 to 30 000 units The injection should be given early in the disease to secure the best results Penicillin should be given as a routine to every case of scarlet fever in order to lower the incidence of complications The dose is 300 000 units of procaine penicillin intra muscularly every 24 hours

## Diphtheria

*(Membranous Croup)*

**Definition** An acute infective disease characterised by localised membrane formation and toxæmic disturbances

**Etiology** Diphtheria is caused by the Coryne bacterium diphtheriæ (B diphtheriæ or Klebs Loeffler bacillus) Three types are described the gravis which ferments starch the intermedius and the mitis which are non fermenters of starch Whether or not a gravis infection produces a severe form of the disease and a mitis infection a mild reaction depends on the resistance of the patient *Predisposing causes* 1 Age Chiefly in children 1 to 15 years 2 Season Autumn and winter epidemics occur 3 Other diseases Scarlet fever and measles 4 Sex Both sexes are equally affected in childhood later females predominate 5 Climate Chiefly in temperate zones

Diphtheria confers only short lived immunity and second attacks were not infrequent The disease is spread by direct contact through droplet infection as in coughing and kissing by fomites especially pencils which are sucked or feeding utensils and handkerchiefs and by milk and carriers The cow s udders may be infected Owing to im

munisation diphtheria has become comparatively rare in Great Britain since 1941 When it does occur, however, it is likely to be as deadly as ever

**Pathology** The membrane is formed of serum fibrin necrosed cellular tissue and organisms It may occur in the pharynx nose larynx, trachea and rarely in the œsophagus and stomach or on the skin Fatty degeneration of the myocardium may develop rapidly Bronchopneumonia is at times due to inhaled diphtheria bacilli The kidneys may show acute interstitial or glomerulonephritis Degeneration of the medullary sheath of peripheral nerves may be found

**Incubation Period** 2 to 10 days, usually 3 to 4 days

**Clinical Findings** There are three main types of the disease faucial laryngeal and nasal, these will be considered separately

*Faucial Diphtheria* The patient is usually a child who feels ill and may have headache or limb pains Frequently there is no complaint of the throat Vomiting or diarrhœa may occur early

*On Examination* The child looks ill and pale in severe cases The exudate in the early stages is seen as isolated greyish yellow patches on the tonsils or fauces These spread and coalesce to form a membrane which may extend to the soft palate pillars of the fauces uvula and posterior wall of the pharynx Swelling of the surrounding tissues in the throat and neck is marked in severe cases and the membrane may become greenish black and emit an offensive odour It can be stripped with some difficulty, leaving a bleeding surface The submaxillary and cervical glands enlarge to a variable degree and may form a collar ("bull neck") The tongue is usually furred The temperature typically is only moderately raised 99 to 101° F, and returns to normal in 3 or 4 days The pulse however is unduly rapid often 110 to 120 The blood There is usually a leucocytosis The blood sugar curve is of the diabetic type in toxic cases The urine Temporary albuminuria is frequent

*Laryngeal Diphtheria* This may occur independently or complicate faucial diphtheria It is rare in adults In children it is a cause of croup' The child becomes hoarse and has paroxysmal attacks of dyspnœa and "croupy" cough

*On Examination* If the laryngeal obstruction is marked signs of deficient pulmonary ventilation are evident such as cyanosis laboured breathing with the accessory respiratory muscles in action intercostal recession, laryngeal excursion and weak air entry into the lungs The membrane may extend to the trachea or main bronchi

*Nasal Diphtheria* This is often associated with faucial diphtheria or may occur independently It is characterised by a watery or mucous nasal discharge which is usually blood stained and often unilateral The membrane is generally in the posterior part of the nose but may be visible in the nostrils

*Other varieties of diphtheria include* Aural usually of the external ear Conjunctival Vulval or anal Cutaneous A membrane may form over wounds or the lesion may resemble eczema or impetigo a diphtheritic whitlow may occur Hæmorrhagic Severe cases of

diphtheria with hæmorrhages into the skin and bleeding from mucous membranes Bacteriological Klebs Loeffler bacilli are found in the throat but no membrane forms

**Differential Diagnosis** *Faucial Diphtheria* This must be differentiated from follicular tonsillitis quinsy, the appearance of the throat after tonsillectomy thrush Vincent s angina agranulocytic angina syphilis of the tonsil scarlet fever mumps and bronchopneumonia

The throat must be examined as a routine in every patient The clinical diagnosis of diphtheria is suggested by the membrane the cervical glandular enlargement the slight pyrexia and the presence at times of albuminuria The bacteriological report does not exclude the disease if no diphtheria bacilli are found and clinical cases should be treated as such without awaiting bacteriological confirmation

*Laryngeal Diphtheria* This must be diagnosed from other causes of croup from the onset of measles or from bronchopneumonia Search should be made for Koplik s spots and a swab should be taken from the throat

*Nasal Diphtheria* This is most likely to be mistaken for nasal discharge from some other cause such as a foreign body impacted in the nose A swab should be taken both from the nose and fauces

**Course and Complications** The course of faucial diphtheria depends very largely on the treatment Serum administered on the first day almost invariably produces rapid improvement if given after the fifth day it has little effect in checking the disease but it should nevertheless be given Laryngeal diphtheria is very serious unless treated early with serum Nasal diphtheria is usually mild but may spread to the fauces and be complicated by paralyses Relapses in diphtheria are rare but the membrane may re form after the third week the relapse is not usually grave

The important complications are *Circulatory Failure* This may be due to (*a*) Toxic myocarditis occurring in the second week or later Clinically the temperature falls the patient becomes restless there may be præcordial pain and vomiting the pulse rate either quickens or slows Variations in the heart sounds are due to gallop rhythm premature systoles partial or complete heart block or bundle branch block auricular flutter or fibrillation The liver may become engorged and the extremities cold About 50% of cases so affected die but those who survive make a complete cardiac recovery (*b*) Peripheral circulatory failure This occurs early during the first week of the illness The patient becomes pale collapsed cold moist and there may be slight general œdema The blood pressure falls to below 100 mm Hg the pulse is rapid and feeble The circulatory failure may be due to (1) a toxic effect on the vasomotor centre (2) peripheral sympathetic paresis (3) a toxic effect on the peripheral vessels and (4) suprarenal hæmorrhage (*c*) Vagal neuritis may be responsible for cardiac failure in some cases

*Allergic Shock* This is due to serum therapy and may occur about the ninth day after its administration There is loss of plasma into the tissue spaces and urticaria This results in a diminished blood

volume and hæmoconcentration In some cases death immediately follows the injection

*Paralyses* (a) Palatal, in the second week There is a nasal voice and nasal regurgitation (b) Ocular in the third and fourth weeks Extrinsic, usually of the VI nerve with squint and diplopia Intrinsic, the pupils usually react to light and accommodation, but there is *difficulty in reading small print, unless the patient is myopic* (c) Pharyngeal in the sixth week There is dysphagia and coughing attacks may occur on swallowing (d) Laryngeal, in the sixth week There is usually adductor paralysis with hoarseness less frequently the abductors are affected, with stridor (e) Diaphragmatic and Intercostal, in the sixth week There is dyspnœa reversal of abdominal movements with respiration intercostal recession and possibly massive collapse of one lung (f) Limbs, Trunk and Neck in the sixth to eighth weeks Weakness of muscles, loss of deep reflexes and blunting of sensation may be found

The paralysed muscles usually recover in about 4 weeks Complete recovery is the rule

Other complications include Bronchopneumonia otitis media mastoiditis nephritis endocarditis with myocarditis cerebral embolus derived from a thrombus in the apex of the left ventricle producing hemiplegia or arterial embolus resulting in gangrene of a limb

**Prognosis** This has been greatly improved by the early use of serum, but 2 to 10% of all cases still prove fatal The prognosis is more unfavourable if the membrane is extensive and in very young children A fall in the diastolic blood pressure is a bad sign The mortality rate is still high for laryngeal and naso faucial diphtheria Hæmorrhagic cases are usually fatal Paralyses are rare in laryngeal diphtheria

**Treatment** *Prophylactic* Diphtheria is a preventable disease and *can be eradicated from a community provided that 80% of children* in the age group of 1 to 15 years are efficiently immunised The susceptibility of individuals can be determined by the Schick test Two tenths of a ml of toxin (1/50 M L D) are injected intradermally and the result is compared with that of the control heated toxin, injected into the other arm Positive reactors can be actively immunised It is advisable to immunise all children irrespective of their Schick reaction It is best done between the ages of 6 and 8 months Combined diphtheria and whooping cough immunisation can be effected as described on p 578 Alternatively Diphtheria Prophylactic A P T (alum precipitated toxoid) can be employed for children The dose under the age of 8 years is two intramuscular injections of 0 5 ml separated by an interval of 1 to 3 months Further injections may be given in doses of 0 2 ml at the age of 3 and 5 years After this 1 ml of Diphtheria Prophylactic T A F (toxin antitoxin floccules) can be injected every 5 years

Diphtheria Prophylactic P T A P (purified toxoid alum precipitated) may be given to children in doses similar to those for A P T It is less liable to cause local reactions For children over the age of 8 years and *for adults* Diphtheria Prophylactic T A F should be used

It is injected intramuscularly in 3 spaced doses of 1 ml The interval between the first and second injection should be 4 weeks and between the second and third 2 to 3 weeks The relationship of prophylactic inoculation to the development of poliomyelitis has been mentioned on p 424 *Quarantine period* Contacts of a case of clinical diphtheria with negative throat swabs and a negative Schick reaction and no sign of diphtheria elsewhere need not be isolated If the Schick reaction is positive the contact must be isolated for 14 days and kept under close observation Immunisation with A P T as described above should be begun If the nose or throat culture is positive the individual may be a carrier of virulent or avirulent bacilli or he may be incubating clinical diphtheria the latter will be determined in a day or so the former can only be decided by inoculation of the organisms into a guinea pig Immunisation with A P T should be carried out at once Prophylactic inoculation of anti diphtheritic serum confers passive immunity for about 3 weeks but it is not justifiable in view of the subsequent danger of anaphylaxis If the contacts are under super vision serum can be given on the first day of the disease should it develop Chronic carriers of virulent bacilli may be treated with stock or autogeneous diphtheria vaccines in doses of 5 to 10 millions every 5 to 7 days or have their tonsils or adenoids removed if they are infected Local penicillin treatment is not successful Ordinary doses of 30 000 units of penicillin every 3 hours do not affect carriers but large doses of 120 000 units every 3 hours may be successful *Isolation period* The patient is isolated for 4 to 6 weeks until the local lesions have healed and there are no discharges and 3 consecutive negative swabs of the nose and throat have been taken at intervals of 3 days

*Curative* The patient must be carried to bed directly the disease is diagnosed and not allowed to walk in view of the possibility of myocardial weakness He must be kept in bed 4 to 8 weeks for the first 2 weeks only one pillow is allowed and the patient is put on absolute rest (see p 566) owing to the danger of heart failure A second pillow is then given and gradually he is allowed to sit up Directly the patient is in bed and a swab has been taken anti diphtheritic serum is injected The amount depends upon the severity of the case and the duration of the disease rather than on the age of the patient A double dose is required if the disease has been present over 48 hours Anaphylactic reactions are uncommon with the purified antitoxin in use A preliminary injection of 0 2 ml should always be given subcutaneously 2 hours before the main injection If there is a local reaction with redness and swelling desensitisation must be effected by injecting intramuscularly doses increasing by 0 5 ml every hour until the amount required has been injected For a mild case affecting the tonsils and fauces 8 000 to 16 000 units are given for an adult or child injected intramuscularly into the middle of the outer side of the thigh Concentrated serum containing 1 500 units per ml or highly concentrated serum containing 2 000 to 2 500 units per ml is obtainable For a moderate case affecting the tonsils soft palate and uvula 24 000 to 40 000 units are given intramuscularly In nasal cases 60 000 units are required Good

effect is shown by improvement in the general condition of the patient who becomes brighter and by cessation of spread of the membrane A second injection should be given in 12 hours if there is no improvement or if the membrane has spread In very severe cases combined intravenous and intramuscular injections of massive doses of serum are given such as 70 000 to 200 000 units The serum is warmed to body temperature but not diluted before intravenous injection and a prophylactic intramuscular injection of liq adrenalin hydrochlor 2 ml, is given at the same time to prevent anaphylactic shock After an intravenous injection, which must be given very slowly a careful watch should be kept on the patient and if the blood pressure falls Pitressin 1 ml (20 units) should be injected intramuscularly Penicillin should be injected intramuscularly in doses of 120 000 units every 3 hours for 2 days This is thought to reduce the number of carriers *A simultaneous intravenous injection of 20 G of dextrose* in 40 ml of normal saline with subcutaneous or intravenous injection of 10 units of insulin is of value in very toxic cases and should be given twice a day for the first two days If intravenous injection *is impossible 20 ml of 10% dextrose may be given intramuscularly* in the buttock Serum sickness, as shown by œdema rashes fever and joint pains is liable to occur about 9 days after the injection but is less frequent in children and with the use of concentrated serum *If it occurs liq adrenalin hydrochlor m 10 should be injected* subcutaneously every 4 hours and antihistamine drugs given by mouth such as Antistin mg 100 tab 1 t i d Benadryl mg 50 capsule 1 t i d, or Thephorin mg 25 tab 1 t i d Calamine lotion may be applied to relieve itching *Local treatment to the throat is dangerous* and should be avoided All discharges should be received on rags and burnt Diet Fluid diet should be given during the febrile period and until the throat is clear when this should be increased to semi solids The bowels should be opened every other day with an enema If the neck is swollen and painful, only warm dry wool should be applied as fomentations may aid the absorption of toxin

*Laryngeal Diphtheria* The minimum dose of antitoxin required for a child or adult is usually *20 000 to 40 000 units* and *much larger* doses are frequently necessary A steam kettle should be kept going day and night For the cough glycerin should be given undiluted in m 60 doses by mouth Tracheotomy is necessary for marked obstruction to breathing *as shown by increasing restlessness* dyspnœa cyanosis tachycardia, and pulsus alternans Laryngeal intubation is coming into fashion again Laryngeal suction is also recommended A semi soft or metal catheter is introduced by means of a direct vision *laryngoscope and its distal end attached to a suction* machine

*Nasal Diphtheria* Serum must be given in large doses for naso faucial diphtheria nasal discharge alone usually ceases after an injection of *60 000 units*

Complications requiring treatment include Toxic myocarditis The patient must be put on absolute rest lying flat with only one

pillow he must not do anything for himself and must be fed washed and attended to lying down The fluid intake should be limited to 2 pints in 24 hours Digitalis is dangerous and may only be used in cases of auricular fibrillation in which there is marked congestive failure Peripheral circulatory failure The patient must lie flat with only one pillow and the foot of the bed raised Liq adrenal hydrochlor m 10 should be injected subcutaneously every 2 to 5 hours The legs and arms should be bandaged Paralysis The affected parts should be rested and tne nuc vom m 10 to 15 t d s given by mouth to an adult and smaller doses for a child If there is pharyngeal paralysis the patient will have to be fed by a nasal tube and pharyngeal mucus should be removed by postural drainage or electric suction to prevent aspiration pneumonia Diaphragmatic paralysis can be treated in the Drinker respirator or Bragg Paul pulsator After convalescence strenuous games should not be played for at least 6 months

## Chicken pox

### (*Varicella*)

**Definition** An acute infectious disease characterised by a vesicular eruption appearing in successive crops and usually fever

**Etiology** The causative organism is a virus Infection is spread by direct contact and possibly slightly by fomites through air or by a third person The virus is presumably inhaled *Predisposing causes* 1 Absence of a previous attack Second attacks are rare 2 Age Children under 10 are chiefly affected 3 Association with a person suffering from herpes zoster Chicken pox occurs at all seasons race and climate play no part in its incidence

**Pathology** The disease is very rarely fatal and has no definite pathology

**Incubation Period** 11 to 21 days usually 14 days

**Clinical Findings** The patient is generally a child but adults are also liable to infection The rash may be the first symptom or there may be an invasion period of 1 or 2 days in which there is malaise headache or shivering

*On Examination* At the onset there is slight pyrexia and prodromal rashes are occasionally seen especially of a scarlatiniform type which may be generalised Prodromal hæmaturia has also been described The true rash appears on the second day and passes rapidly from macule to papule and then to the typical vesicular eruption

The vesicles become pustular before they burst in about 24 hours time The rash comes out in crops for 2 to 7 days so that spots in the various stages are seen simultaneously in close proximity The eruption first appears on the trunk and spreads centrifugally to the face scalp and upper parts of the limbs The legs forearms hands and feet are involved to a lesser degree and the spots may be seen in the axilla and on the palate (see Fig 54)

The vesicles are oval of varying size averaging about 1 by 3 mm with a small red areola They feel smooth and are superficial On the

scalp, hands and feet they are deeper and more shotty Usually they are not umbilicated and collapse on puncture The vesicles are generally discrete but a few may coalesce The crusts take from 4 days to 4 weeks to separate and leave white scars which last for many years The skin usually irritates intensely when the rash is profuse The temperature This is variable and usually slight but in a severe case may be considerably raised for a few days, not falling as the rash comes out (see Fig 55) The blood There is a lymphocytosis for a few days while the rash is out

*Varieties* 1 *Hæmorrhagic* Hæmorrhages appear and there may

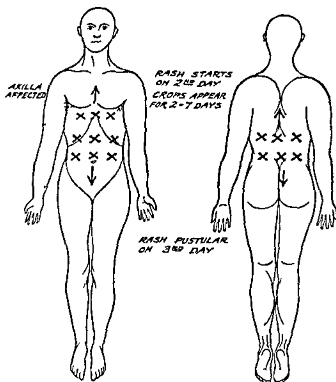



FIG 54 DIAGRAM OF RASH IN CHICKEN POX
The crosses indicate the usual site of the first appearance of the rash

also be bleeding from the mucous membranes This is very rare but it is not necessarily fatal 2 *Gangrenous* There is necrosis of the skin and subcutaneous tissues It is met with in debilitated children and is usually fatal

**Differential Diagnosis** This necessitates a consideration of the possibility of small pox or of skin diseases such as impetigo scabies lichen urticatus pemphigus and generalised herpes and of scarlet fever in the invasion stage The chief distinguishing features of chicken pox are the absence of severe symptoms during the invasion period the distribution of the rash its appearance in crops and the absence of a secondary temperature (see also small pox p 572) Careful attention

to the character of the lesions usually serves to distinguish other skin conditions If lesions are found in the mouth, it is strongly in favour of chicken pox

Herpes zoster is now often thought to be caused by the same virus as that which produces chicken pox perhaps it acts through the nervous system in the former and through the blood in the latter Its distribution is related to cutaneous nerve areas

**Course and Complications** Varicella usually pursues a mild course Complications are very infrequent but include nephritis laryngitis bronchopneumonia encephalomyelitis conjunctivitis and otitis media Encephalomyelitis is characterised by tremors ataxia or by signs of transverse myelitis It may occur during the second week of the illness and be heralded by fever vomiting or a rigor Varicella is more frequently associated with scarlet fever than with any other acute infection

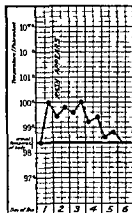


Fig 5 Temperature Chart in Chicken pox

**Prognosis** This is very good except in the severe types of the disease Recovery usually occurs in cases of encephalomyelitis

**Treatment** *Prophylactic* Inoculation with convalescent serum has only proved moderately effective *Quarantine period* This is 21 days but the contacts need not be isolated for the first week *Isolation period* The patient must be isolated until the last scab has separated which usually takes 3 to 4 weeks

*Curative* The patient must be put to bed until the temperature is normal, fresh spots have ceased to appear and all spots have reached the crusting stage Usually the only treatment required beyond that necessary for any mild feverish illness is a local application for the irritating skin The patient should not be allowed to scratch the spots A dusting powder of equal parts of talc zinc oxide and borax may be applied and baths given when the crusts form with warm water coloured pink with potassium permanganate In cases with secondary infection of the lesions crystalline penicillin should be given in doses of 60 000 units intramuscularly every 12 hours A swab should al o be taken to exclude the presence of C diphtheriæ Zinc ointment (ung zinc oxid B P ) is applied to the sores left after the crusts have separated

## Small pox
## (*Variola*)

**Definition** An acute infectious disease characterised by fever and a typical rash

**Etiology** Paschen bodies which are present in the fluid of the pocks are probably the causative virus Guarnieri bodies which are found in the epithelial cells of the pocks of small pox are considered to be agminations of Paschen bodies Infection is spread directly

scalp, hands and feet they are deeper and more shotty Usually they are not umbilicated and collapse on puncture The vesicles are generally discrete but a few may coalesce The crusts take from 4 days to 4 weeks to separate and leave white scars which last for many years The skin usually irritates intensely when the rash is profuse The temperature This is variable and usually slight but in a severe case may be considerably raised for a few days, not falling as the rash comes out (see Fig 54) The blood There is a lymphocytosis for a few days while the rash is out

*Varieties* 1 *Hæmorrhagic* Hæmorrhages appear and there may

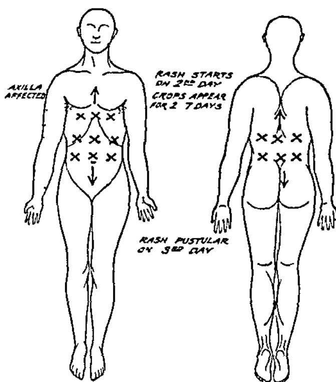



Fig 54 Diagram of Rash in Chicken pox
The crosses indicate the usual site of the first appearance of the rash

also be bleeding from the mucous membranes This is very rare but it *is not* necessarily fatal 2 *Gangrenous* There is necrosis of the skin and subcutaneous tissues It is met with in debilitated children and is usually fatal

**Differential Diagnosis** This necessitates a consideration of the possibility of small pox or of skin diseases such as impetigo scabies lichen urticatus pemphigus and generalised herpes and of scarlet fever in the invasion stage The chief distinguishing features of chicken pox are the absence of severe symptoms during the invasion period the distribution of the rash its appearance in crops and the absence of a secondary temperature (see also small pox, p 572) Careful attention

to the character of the lesions usually serves to distinguish other skin conditions If lesions are found in the mouth, it is strongly in favour of chicken pox

Herpes zoster is now often thought to be caused by the same virus as that which produces chicken pox perhaps it acts through the nervous system in the former and through the blood in the latter Its distribution is related to cutaneous nerve areas

**Course and Complications** Varicella usually pursues a mild course Complications are very infrequent but include nephritis laryngitis bronchopneumonia encephalomyelitis conjunctivitis and otitis media Encephalomyelitis is characterised by tremors ataxia or by signs of transverse myelitis It may occur during the second week of the illness and be heralded by fever vomiting or a rigor Varicella is more frequently associated with scarlet fever than with any other acute infection

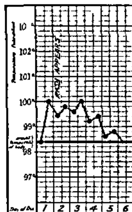


Fig 55 Temperature Chart in Chicken pox

**Prognosis** This is very good except in the severe types of the disease Recovery usually occurs in cases of encephalomyelitis

**Treatment** *Prophylactic* Inoculation with convalescent serum has only proved moderately effective *Quarantine period* This is 21 days but the contacts need not be isolated for the first week *Isolation period* The patient must be isolated until the last scab has separated which usually takes 3 to 4 weeks

*Curative* The patient must be put to bed until the temperature is normal fresh spots have ceased to appear and all spots have reached the crusting stage Usually the only treatment required beyond that necessary for any mild feverish illness is a local application for the irritating skin The patient should not be allowed to scratch the spots A dusting powder of equal parts of talc zinc oxide and borax may be applied and baths given when the crusts form with warm water coloured pink with potassium permanganate In cases with secondary infection of the lesions crystalline penicillin should be given in doses of 60 000 units intramuscularly every 12 hours A swab should also be taken to exclude the presence of C diphtheriæ Zinc ointment (ung zinc oxid B P ) is applied to the sores left after the crusts have separated

## Small pox
## (*Variola*)

**Definition** An acute infectious disease characterised by fever and a typical rash

**Etiology** Paschen bodies which are present in the fluid of the pocks are probably the causative virus Guarnieri bodies which are found in the epithelial cells of the pocks of small pox are considered to be agminations of Paschen bodies Infection is spread directly

by fomites by third persons and probably by flies the virus being inhaled *Predisposing causes* 1 Absence of protection by vaccination 2 Absence of a previous attack 3 Season Epidemics tend to occur in the winter and spring 4 Race and climate Small pox is more common in the tropics especially where the disease has not been endemic 5 Age and sex Earlier epidemics, such as those of 1788–1800 and 1837–40, affected chiefly children under 5 years now youths and adults of either sex are attacked

**Pathology** At autopsy eruptive lesions may be seen in the

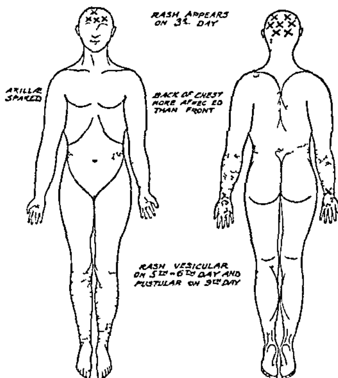



FIG 56 DIAGRAM OF RASH IN SMALL POX

Peripheral extensor surfaces of limbs especially involved Rash profuse on pressure points such as waist or garter line The crosses indicate the usual site of the first appearance of the rash

œsophagus, larynx and trachea Hæmorrhage may occur in the muscles and solid organs Small yellow areas of focal necrosis are often found in the testicles The spleen may be enlarged and soft

**Incubation Period** 10 to 14 days usually 12 days

**Clinical Findings** The patient may be a child or an adult He is suddenly taken ill with shivering or a rigor headache and backache Giddiness nausea vomiting and severe malaise are often present

*On Examination* During the invasion period the patient looks tired and the temperature is often raised during the first 2 days to 102 or 104 F *Prodromal rashes* may be seen these are 1 Petechial involving especially the bathing drawers area (triangular rash) There

are small bright or dark red spots which may also be found in the axillæ 2 Erythematous either scarlatiniform morbilliform or multiform of a wider distribution The spleen may be palpable The true rash appears on the third day and is maculo papular, it becomes vesicular *by the fifth or sixth day and pustular by the ninth day* The pustules burst about the twelfth day with crust formation by the sixteenth day The macules first appear on the forehead scalp and back of the wrists and spread over the face and peripheral portions of the limbs to the trunk The rash involves especially extensor surfaces exposed parts and those subject to pressure The axillæ are usually spared It is more scanty on the abdomen loins chest neck and flexures of the limbs The legs are less involved than the arms and usually the upper half of the limbs are less involved than the lower

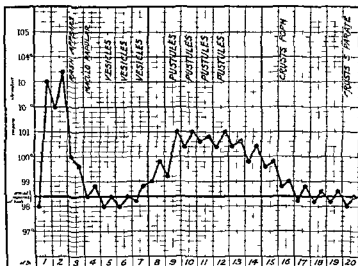



FIG 57 TEMPERATURE CHART IN SMALL POX

The eruption is also seen on the palms soles conjunctivæ and in the mouth (see Fig 56)

The macules vary in size are dark red and rapidly become papular firm and shotty The vesicles are pearl coloured and loculated so that they do not collapse on puncture Umbilication due to depression of the centre by a sweat duct or hair follicle *is often seen The pustules* are yellowish with a red areola and the skin around may be œdematous The brownish black crusts formed after rupture usually separate in from 2 to 8 weeks leaving depressed scars which are often permanent

There is much irritation and the skin may become tense and painful with a very offensive odour during the pustular stage The temperature usually falls to about 99 F or lower as the true rash appears and rises again (secondary fever) with pustulation to about 101 F It then *gradually falls to normal* about the middle of the third week (see Fig 57) During the acute stage the patient is very prostrated

fomites by third persons and probably by flies, the virus being [...]led. *Predisposing causes* 1 Absence of protection by vaccination. [A]bsence of a previous attack. 3 Season Epidemics tend to occur [in t]he winter and spring 4 Race and climate Small pox is [com]mon in the tropics, especially where the disease has not been endemic. [A]ge and sex Earlier epidemics, such as those of 1783–1800 and [...]0 affected chiefly children under 5 years, now youths and adults [of ei]ther sex are attacked.

Pathology At autopsy eruptive lesions may be seen in the

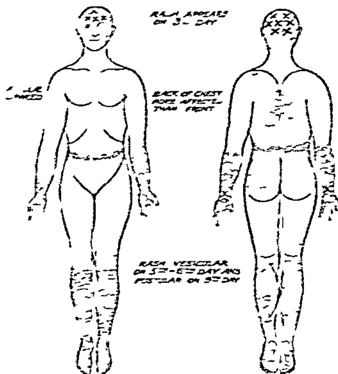



FIG. 26. DIAGRAM OF RASH IN SMALL-POX.
Peripheral extensor surfaces of limbs especially involved. Rash profuse on pressure points such as waist or garter line. The crosses indicate the usual site of the first appearance of the rash.

[oeso]phagus, larynx and trachea. Hæmorrhage may occur in the [musc]les and solid organs. Small yellow areas of focal necrosis are [...] found in the testicles. The spleen may be enlarged and soft.

[In]cubation Period. 10 to 14 days, usually 12 days.

[C]linical Findings. The patient may be a child or an adult. He is [sudd]enly taken ill with shivering or a rigor headache and backache. [...]ness, nausea, vomiting and severe malaise are often present.

[...]n *Examination* During the invasion period, the patient looks [...], and the temperature is often raised during the first 2 days, to [...]or 104° F *Prodromal rashes* may be seen these are 1. Petechial, [...]ving especially the bathing-drawers area (triangular rash). There

**Course and Complications** The course of a case of average severity been described above Confluent or hæmorrhagic cases are much e severe and usually rapidly fatal. The complications include ngitis bronchitis, bronchopneumonia heart failure conjunctivitis neal ulceration, panophthalmitis, otitis media, mastoiditis, orchitis, sores, boils encephalomyelitis neuritis and osteomyelitis

**Prognosis** This varies with the protection afforded by previous cination and the type of disease The most critical days are usually twelfth to the fourteenth If the initial fever is slight the prognosis sually good but the converse is not true.

**Treatment.** *Prophylactic* Since the repeal of the vaccination acts Great Britain in 1948 only about 25% of infants in England and les are vaccinated in the first year of life Primary vaccination is st successful between the third and sixth months Primary vac ation at school age is discouraged owing to the risk of encephalo elitis Vaccination successfully performed in infancy affords tection for about 7 years It should be repeated at the ages of 7 14 l 21 and again if there is an epidemic Chicken pox should be made ifiable during an epidemic of small pox The patient s house and contents should be disinfected *Quarantine period* Contacts should vaccinated and kept under observation for 16 days *Isolation period* e patient must be removed to and kept in an isolation hospital il all the scabs have separated and the skin has healed usually for reeks or longer

*Curative* The patient is kept in bed in a well ventilated room and ght light *is excluded Crystalline penicillin should be given at the* set of the disease 500 000 units being injected intramuscularly every hours Diet Milk and fluids are given during the febrile period of disease the diet being rapidly increased when the temperature falls llet The hair should be cut short and the skin sponged twice daily h warm water A freshly prepared saturated solution of potassium manganate should be applied all over the skin on beginning treat nt this can be repeated in a day or so with a 1% solution Warm ths to which potassium permanganate is added to make a pale ik colour are welcomed after the pocks have burst The virus is stroyed by 1 in 10 000 permanganate The face should be covered th a lint mask soaked in iced water and covered with oiled silk The ensive odour can be mitigated by dabbing the skin with 1 in 100 rbolic acid solution instead of the permanganate The eyelids should smeared with vaseline and the eyes frequently bathed with boracic ion If any eruption appears inside the lids they should be everted d treated with vaseline containing hydrarg oxid flav gr 8 and ropin sulph gr 16 to the oz daily If there are signs of further flammation 10% Albucid drops should be used every 4 hours and cal heat applied frequently

To aid the separation of the crusts especially from the face a thin yer of linseed poultice *may be applied on a lint mask* and changed ery 2 hours When the crusts have separated zinc ointment (ung nc oxid B P ) can be applied to the scars

and insomnia or delirium may be marked The blood Ther leucopenia during the first 5 days and leucocytosis during the sec and third weeks with the lymphocytes forming about 40% of count The urine Febrile albuminuria is often present Labora tests of diagnostic aid include the complement fixation test using contents of the vesicles as the antigen, with serum from sensit rabbits The virus may also be isolated from the vesicular fluid growing it on the chorioallantois of the chick embryo Further sm from the base of a vesicle or papule on a slide fixed with alcohol stained with methyl violet, show numbers of 'elementary bodi allowing a presumptive diagnosis to be made in the papular stage

*Varieties* 1 *Discrete* A mild type 2 *Semi-confluent* W little normal skin between the spots 3 *Confluent* The pustules f and there is much subcutaneous œdema (These types are judged the rash on the face and forearms and they are all varieties of Vari vera ) 4 *Varioloid* (abortive) Slight cases, often modified owing previous vaccination There may be no rash (variola sine eruptio 5 *Variola Minor* (*paravariola*) *Alastrim and Amaas* Mild types occ ring in Brazil and Africa, which have been reported in England si 1919, and were originally described in Gloucestershire by Jenner Th is no secondary fever and the rash may come out in crops Adults chiefly affected 6 *Hæmorrhagic* Two types are described both v malignant (*a*) Purpuric (black small pox) with very scanty ra Hæmorrhages occur in the skin, mucous membranes and inter organs (*b*) Variola hæmorrhagica pustulosa in which hæmorrha occur after the development of the rash which may not reach t pustular stage Small hæmorrhages into the pocks do not constitute tr hæmorrhagic small pox and are not necessarily severe 7 *Congenita*

Differential Diagnosis In the invasion stage small pox may mistaken for influenza pneumonia, scarlet fever, measles or purpur Search should be made for the petechial rash described abov The typical features of the onset of measles and scarlet fever a absent

When the rash has appeared the differential diagnosis include Chicken pox measles typhus fever pustular drug eruptions such a those caused by iodides and bromides a pustular syphilide and acn The distribution of the rash is most important The greatest difficult usually occurs in the case of chicken pox In small pox the rash is mo profuse on the face and extremities and in chicken pox on the trunk The axilla is usually free from eruption in small pox whereas this are is affected in chicken pox In small pox the rash does not come ou in crops as it does in chicken pox In small pox the typical vesicle i round and rather deep whereas it is oval and superficial in chicken pox Small pox is the only infectious disease in which the temperatur falls with the appearance of the eruption As mentioned above smear made from the papules are of diagnostic value

If the patient has recently been successfully vaccinated it is improb able that he is suffering from small pox If a successful test vaccinatio is made on the second day of illness, small pox is practically excluded

temperature is raised 2 Generalised vaccinia This may be due to (a) auto-inoculation by scratching the vesicles occurring elsewhere as on the face (b) blood borne infection generalised papules and vesicles forming between the fourth and tenth days 3 Protein rashes Various erythemata may occur 4 Encephalomyelitis A dread but rare complication proving fatal in about 35% of cases it has been specially noted since 1923 and occurs usually in children and young adults who have not been previously vaccinated It is possibly due to a dormant virus activated by the vaccine virus The symptoms are noted about the tenth or twelfth day after vaccination The patient is drowsy complains of headache vomits and becomes delirious with various pareses Post mortem there is perivascular demyelination in the brain and cord Treatment consists in the intravenous or intrathecal injection of 5 to 30 ml of serum taken from an individual who has been successfully vaccinated 14 days previously

## Mumps

### (*Epidemic Parotitis*)

Definition An acute infectious disease characterised by enlargement of the salivary glands usually the parotids

Etiology The virus is present in the saliva and spread by droplets *By some it is considered to be neurotropic the nervous system being* primarily involved with secondary affection of the salivary glands by others it is thought to be a septicæmia the virus having a predilection for the salivary glands The patient is considered to be infectious for 2 or 3 days before the swelling appears *Predisposing causes* 1 Age 5 to 15 years and again 18 to 25 years 2 Sex Chiefly males 3 Season Winter and spring 4 Absence of a previous attack Second attacks are rare 5 Overcrowding in army huts Epidemics occur all over the world

Pathology The parotid swelling is chiefly due to hyperæmia and œdema of the connective tissue Fibrosis of the testis with atrophy of the glandular epithelium may occur The pancreas may be hyperæmic

*Incubation Period. 12 to 23 days usually 18 days*

Clinical Findings The patient is usually a boy or young adult who complains of headache malaise sore throat nose bleeding or *stiff* neck before he notices the pain and swelling in one parotid region Often however the parotid swelling is the first symptom

*On Examination* Some fulness is seen behind the angle of the jaw this spreads forward over the masseter and down into the neck and the parotid rapidly becomes definitely enlarged In a day or so the opposite gland is usually affected the skin becomes tense over the gland and the jaw can only be opened slightly owing to pain Trismus may be noted and salivation is usually increased *More rarely the* submaxillary or sublingual glands alone are affected and here too there is usually a bilateral spread When the parotid is involved the *orifice of Stenson s duct in the* mouth is usually seen to be swollen Rarely the lachrymal glands enlarge The spleen may be just palpable The temperature

To relieve the severe pain in the back and the headaches phenacetin gr 7 or aspirin gr 10 may be given To induce sleep, Dover s powder (pulv ipecac et opii B P ) gr 10 or paraldehyde m 60 to 120 may be required Delirium is best relieved by the injection of hyoscin hydro brom gr 1/100 A steam inhalation containing tinc benzoin co m 60 to 1 pint relieves the laryngitis The mouth should be cleaned after each feed with a swab dipped in glycerin thymol co (B P C ) m 60 to 5 oz of water

## Vaccinia

*(Cow pox)*

**Definition** This is a disease of cows but the term also includes the effects of inoculation of man with vaccine lymph containing the attenuated variola virus

**Vaccination** The lymph now used in Great Britain is derived from sheep Lymph will remain potent when kept in a domestic refrigerator for 2 weeks If kept in the dark and in a cool place it should be active for about 7 days

The multiple pressure method is usually recommended The skin over the posterior border of the deltoid should be washed with soap and water and dried The Hagedorn needle should be sterilised by heat and allowed to cool A drop of lymph is placed on the skin The needle is held at an angle of 45 with the skin and the side of the point of the needle is pressed through the drop of lymph with sufficient force to indent the skin and enter its superficial layers but not to draw blood This pressure movement is repeated 30 times for revaccination and also for a primary vaccination when it is urgent or obligatory For non urgent primary vaccination 20 pressures are satisfactory Alternatively vaccination may be made by a single linear scratch about half an inch long through a drop of lymph The scratch should be sufficiently deep to leave a red mark but not to draw blood Primary vaccination in infants requires no dressing before the fifth or sixth day when a loose application of sterile gauze should be applied A special dressing can be obtained for adults which protects the crust and allows free passage of air, so that the lesion quickly heals The vaccination should be inspected on the seventh day If a primary vaccination takes a papule forms on the third day, which is vesicular by the fifth day, continues to enlarge until the eighth day and maturates becoming pustular by the tenth day The vesicle dries and forms a scab, which separates between the fourteenth and twenty first days The skin around the vesicle is red and swollen during the second week and the axillary glands are usually enlarged A scar results which shows small depressions or foveations During the second week there is usually malaise and some pyrexia Vaccination takes" less each time and often not at all after the third time Vaccination is contraindicated in the presence of any chronic skin lesion such as eczema in acute infections or if the child has recently been exposed to an infectious disease

**Complications** These include 1 Sepsis The arm may become red and brawny and very painful the axillary glands enlarge and the

in doses of mg 1 4 times a day may prevent orchitis, or relieve the symptoms if it occurs Operation is not required for pancreatitis

## Whooping Cough

### (*Pertussis*)

**Definition** An acute infective disease characterised by paroxysms of coughing followed by an inspiratory whoop

**Etiology** Whooping cough is caused by the Hæmophilus pertussis (B pertussis or the Bordet-Gengou bacillus) *Predisposing causes* 1 Age Chiefly children between 2 and 5 years but adults are affected 2 Sex Females predominate slightly 3 Season March and April especially The disease is spread by droplet infection rarely by fomites or a third person It is most infectious in the catarrhal stages Second attacks may occur

**Pathology** Whooping cough is rarely fatal apart from complications such as bronchopneumonia The trachea larynx and bronchi show catarrhal changes and the bronchial glands are enlarged The bacillus is found especially in the larynx and trachea

**Incubation Period** 6 to 18 days usually 7 days

**Clinical Findings** The disease is very common about 65% of children in Great Britain suffering from it before the age of 16 years *The catarrhal stage* The patient is generally a child who first shows the symptoms of a severe cold and a little cough The temperature is slightly raised 99° to 100° F The blood There is a leucocytosis of 12 000 to 27 000 per c mm with 60% or more lymphocytes If the patient coughs over a blood agar potato plate the H pertussis can often be isolated The organism may also be isolated by pernasal or postnasal swabbing The former method is the more satisfactory *The paroxysmal stage* This begins about 5 to 14 days later It is characterised by a noisy rapidly repeated explosive cough during which the child is cyanosed tears may run from the eyes and mucus from the nose This is immediately followed by a long drawn crowing inspiration or whoop The attacks tend to be provoked by feeding by exertion or emotions but they also occur at night After the attack the child may drop asleep She is often alarmed by the paroxysm and may get out of bed or run to her mother Vomiting may occur at the end of the attack and the patient lose weight In the intervals the conjunctivæ tend to be congested and the face a little swollen ulceration of the frænum linguæ may be seen due to friction against the teeth during the attacks The temperature is usually normal The blood still shows a leucocytosis The urine may contain an excess of uric acid

In some cases the attack is mild or abortive and although paroxysms of barking cough occur by day and night there is no whoop *The convalescent stage* After about 4 to 6 weeks the whoop disappears and the cough gradually lessens although it may still be provoked by running or sea bathing The leucocyte count has now returned to normal

**Differential Diagnosis** Whooping cough must be diagnosed from

rises usually to 101° F or over and takes 3 or 4 days before it reaches normal With complications such as orchitis or pancreatitis it rises again Occasionally orchitis, pancreatitis or benign aseptic meningitis may occur without parotitis The pulse is usually slow, 50 to 60 The blood There is a lymphocytosis, the average figure being 48% and the total white cell count is increased The urine Albuminuria occurs in about one third of cases The cerebro spinal fluid An excess of lymphocytes and of globulin is frequently found, apart from meningitis

**Differential Diagnosis** This is not usually difficult If the parotid is enlarged other causes such as sarcoidosis sepsis drugs such as iodides tumours and Mikulicz s disease must be excluded The lungs should be X rayed in suspected sarcoidosis to see if any characteristic changes are present (see p 608) Septic parotitis is usually unilateral and often suppurates In Mikulicz s disease the parotid enlargement is chronic painless and the lachrymal glands are also affected Cervical adenitis must also be excluded such as that due to diphtheria or glandular fever In a case of mumps in which salivary glands other than the parotid are involved the glands are usually affected bilaterally in a day or so The lymphocytosis in mumps also aids in the diagnosis

**Course and Complications** The swelling in mumps usually disappears in less than a week and the glands practically never suppurate The most frequent complication is orchitis which is however rare before puberty It occurs usually about the seventh day or later generally only one testis is affected Hydrocele and œdema of the scrotum may also develop In females tenderness may occur over the ovary Other complications include myocarditis acute pancreatitis as evidenced by epigastric pain vomiting and constipation and a high serum amylase content mastitis otitis media labyrinthitis and nerve deafness, peripheral neuritis and neuritis of the II VII VIII and III cranial nerves Meningo encephalitis meningism or meningitis may occur at any stage in the illness

**Prognosis** This is very good Permanent sterility, but rarely impotence may result from orchitis permanent deafness or diabetes mellitus are rare sequelæ

**Treatment** *Prophylactic* It is very doubtful whether convalescent serum or gamma globulin has any prophylactive or curative effect in mumps *Quarantine period* This is 26 days but children may attend school for the first week *Isolation period* The patient should be isolated for a week after all swelling has gone from the glands and for a minimum of 14 days

*Curative* The patient should be in bed for 10 days as the recumbent position lessens the liability to orchitis Fluid diet is required until mastication is painless The bowels should be opened with aperients such as salines or cascara sagrada Mouth washes should be used frequently such as glycerin thymol co (B P C ) Glycerin belladon (B P C ) should be painted over the swollen glands and fomentations applied if there is much pain For orchitis the affected testicle is supported with a suspensory bandage or small pillow, and glycerin belladon (B P C ) with fomentations are applied Stilbœstrol

is severe the feeds should be given about 10 minutes after the vomit as this offers the best chance of the nourishment being retained Dextrose gr 60 should be given t i d to prevent acidosis Discharges and sputum should be burnt An inhalation of tnc benzoin co m 60 to 1 pint of steaming water often relieves catarrh For the cough a sedative mixture containing Tnc opii camph m 5 tnc ipecac m 3 syr pruni serot m 10 aq chlorof ad m 120 should be given six hourly for a child of 5 If the respiration rate is raised the child should be nursed in an oxygen tent Good results have been obtained with aureomycin in doses of 25 mg /lb body weight divided into 4 doses daily for a week It is also effective against secondary infections If the paroxysms still continue a rectal injection of ether m 60 and olive oil fl oz ½ may be given twice daily or phenobarbitone gr 1/24 t i d in milk for an infant Gamma globulin obtained from hyperimmune serum may be injected intramuscularly in doses of 2 5 to 5 ml on four successive days Convulsions in an infant should be treated with hot baths lumbar puncture inhalations of chloroform the rectal injection of pot brom gr 6 in water fl oz 1 or the subcutaneous injection of gr 1/8 to gr 1/16 of sodium phenobarbitone

## Typhoid Fever

### (*Enteric Fever*)

**Definition** An acute infective disease characterised by continued fever enlargement of the spleen bacillæmia involvement of intestinal lymphatic tissue and usually a roseolar eruption

**Etiology** Typhoid fever is caused by the bacterium typhosum (Salmonella typhosa ) *Predisposing causes* 1 Age Chiefly between 5 and 35 years 2 Sex Slight excess of males 3 Season Chiefly the autumn 4 Overcrowding Especially in tents in the army 5 Bad sanitation 6 Absence of a previous attack or recent inoculation The disease occurs all over the world It is spread by patients and carriers through contamination of food and water especially milk cream butter oysters ice cream and watercress it is also spread by flies (their feet vomit and excreta) by fomites and by inhalation of bacilli as from dried excreta The nurse in charge of a patient may contract typhoid fever by the latter method Epidemics are usually due to a carrier contaminating the food supply of an area owing to bacilli on his fingers soiled with urine or fæces or to sewage contamination of drinking water

**Pathology** Infection probably spreads from the bowel to the blood the organisms are then excreted by the bowel or kidneys The bacilli tend to lodge in the gall bladder and intestinal lymphoid tissue and they may be found in the cutaneous spots Intestinal lesions include swelling of Peyer s patches and solitary lymphatic follicles the last 18 inches of the ileum are chiefly involved These areas may ulcerate and perforate The spleen is enlarged and soft the mesenteric glands are enlarged the skeletal muscles may undergo Zencker s degeneration

**Incubation Period.** 7 to 21 days usually 14 days

**Clinical Findings** The onset is usually insidious the patient

other catarrhal infections such as a cold the early stages of measles and laryngeal diphtheria, from paroxysmal coughs due to tracheo bronchitis or enlarged bronchial glands, which in children may be tuberculous and from other causes of mediastinal pressure in adults If there is no whoop the diagnosis depends upon the character of the cough, the blood count and isolation of the bacillus from droplets or sputum

**Course and Complications** The usual course is as described above The most important complications are Bronchitis bronchopneumonia pulmonary collapse from blockage of a bronchus with mucus encephalitis convulsions and cerebral hæmorrhage Other complications include spasm of the glottis bronchiolectasis, acute emphysema surgical emphysema, spontaneous pneumothorax otitis media, prolonged vomiting, bleeding from the nose ears, eyes and gums The cough may provoke hernia or prolapse of the rectum Children in hospitals and school sanatoria are very liable to contract diphtheria and a prophylactic injection of 1 000 to 2 000 units of diphtheria antitoxin is advisable Bronchiectasis or tuberculosis may supervene Convulsions may lead to mental deficiency

**Prognosis** The disease is serious in very young infants in debilitated and rickety children, and in association with convulsions and bronchopneumonia In an uncomplicated case the severity may be gauged by the number of paroxysms any figure over 20 in the 24 hours being grave The outlook is bad where there is inability to retain food or when there are frequent convulsions

**Treatment** *Prophylactic* Infants can be simultaneously immunised against diphtheria and whooping cough by the use of Diphtheria Pertussis Prophylactic This should be given between the ages of 6 months and 1 year the dose being 1 ml injected intramuscularly, repeated twice at an interval of a month, and again during the second year and then every 2 years until the age of 6 or 7 Alternatively infants and children up to the age of 8 years should be immunised with H pertussis vaccine made from killed Phase I strains The first injection is best given about the age of 12 months after diphtheria immunisation has been successfully carried out The dosage is 5 000 millions repeated after a week then 10 000 millions after a month and again a month later A further injection should be given a year later or when the child is first exposed to whooping cough If there is a severe local reaction or if convulsions occur no further vaccine should be given Vaccination should not be made during an epidemic of poliomyelitis if there is a family history of organic nerve disease, or if the child has recently been exposed to an infectious fever Gamma globulin prepared from hyperimmune serum 2 5 to 5 ml intramuscularly will afford temporary immunity to an unvaccinated contact *Quarantine period* This is 3 weeks *Isolation period* This is 6 weeks

*Curative* The patient should be in bed during the catarrhal stage and until the vomiting becomes infrequent The room should be well ventilated but maintained at a temperature of 60° F Diet Liquids and semi solids are advisable until the paroxysms lessen When vomiting

falls to normal in the mornings rising a little in the evenings The abdominal reflex reappears the spleen is no longer palpable and the general condition improves During this week repair takes place in the areas where the sloughs have separated Recrudescences may occur the temperature rising irregularly, but the spleen does not enlarge and no fresh spots appear *Relapses* After the temperature has been normal for about a week it may rise again in step-ladder form and the disease be repeated, with fresh spots enlargement of the spleen and intestinal symptoms The duration of the relapse is usually shorter than that of the original disease *Varieties* 1 *Mild* (including ambulant ' cases) 2 *Apyrexial* 3 *Severe* (*a*) Hæmorrhagic (*b*) Pneumo typhoid the disease starts with lobar consolidation of the lung (*c*) Nephro-typhoid the onset is with symptoms of acute nephritis (*d*) Meningo typhoid the onset is with symptoms of meningitis

**Differential Diagnosis** At the onset typhoid fever may be mistaken for influenza gastro enteritis pneumonia nephritis or meningitis The

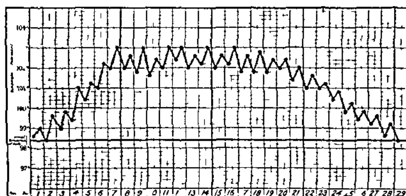



FIG 58 TEMPERATURE CHART IN TYPHOID FEVER

temperature should always be taken as ambulant cases will then be kept under observation when found to be pyrexial The continued fever may be confused with that due to paratyphoid fever tuberculous peritonitis miliary tuberculosis pyelitis undulant fever infective endocarditis or Hodgkin s disease Typhus fever and secondary syphilis have also caused errors of diagnosis A typical clinical picture is not always present and reliance must be placed on blood and fæces culture and the Widal test A person inoculated with T A B vaccine will give a positive Widal reaction for a year or more but if an active typhoid infection supervenes the agglutinin titre of the serum usually waxes and wanes during the course of the disease Virulent strains of typhoid bacilli contain two varieties of somatic antigens O and Vi the third antigen H which is flagellar is not an indication of active infection These antigens give rise to corresponding agglutinins in the serum Agglutination of the H form is probably indicative of previous inoculation whereas O agglutination signifies active infection

**Course and Complications** The typical course has been described

complaining of lassitude, frontal headache, backache, constipation anorexia epistaxis, malaise and insomnia associated with a gradually rising temperature In some cases there is a sudden onset with fever, vomiting, rigors, and delirium As the disease runs an average course of 4 weeks, the main features during each week will be considered

*First Week* (Invasion stage or advance) The patient may be up (ambulatory typhoid) the face is a little *flushed* and the tongue has a white fur with the edges and tip clean The pupils are *dilated* There may be complaint of abdominal pains or discomfort The temperature rises in a step ladder fashion with a progressive evening rise and it drops ½ to 1 degree each *morning* In this way by the end of the week it may reach 102° to 103° F (see Fig 58) The *pulse* is relatively slow, 90 to 100, and often dicrotic The *abdomen* is *a little* swollen owing to flatulence, and there may be gurgling on palpation over the cæcum The abdominal reflex is usually absent The spleen *is just palpable* Typhoid spots (rose spots) may appear about the seventh day pink macules, 2 to 4 mm in diameter *fading on pressure* and situated on the trunk and abdomen They are not *numerous* perhaps 6 to 12 they disappear in 2 to 4 days, and fresh crops appear There is often a little bronchitis The blood There is leucopenia 4,000 to 5 000 white cells per c mm At times there is a slight *leucocytosis* during the first week Typhoid bacilli are present The urine Febrile albuminuria may occur Typhoid bacilli may be present at the end of the week but more often not until the second week. The motions may be costive or loose ( pea soup ), yellowish and offensive Typhoid bacilli may be present but they are found in a higher percentage of cases in the second and third weeks During this time there may be swelling of Peyer s patches

*Second Week* (Fastigium) This stage has largely been abolished by the use of chloramphenicol The patient is more prostrated the headache is less marked and deafness may develop The tongue is drier and coated in the centre with a clean tip and edges Insomnia may now be troublesome and delirium occur The temperature remains sustained at about 101° to 103° F and the pulse accelerates a little to just over 100 The blood The Widal reaction becomes positive after the tenth day typhoid bacilli are usually present The bowels There *is a greater* tendency to diarrhœa during this period During this week sloughs may form in Peyer s patches

*The Third Week* (Defervescence) The patient is still more exhausted and may sink into the typhoid state ' with delirium muscular twitching coma vigil, a dry and shiny tongue and sordes on the lips This stage of wasting is seldom seen now as adequate nourishment is supplied It is the danger period owing to the risk of hæmorrhage or perforation During this week the intestinal sloughs may separate Usually there is however improvement towards the end of this week and the temperature begins to fall by lysis rarely by crisis The abdomen may be more distended by meteorism and the patient is often much wasted

*The Fourth Week* (Convalescence) The temperature gradually

*Curative* The patient is put to bed and kept there until the tempera ture has been normal for 2 weeks The course of the disease has been much modified by the use of chloramphenicol The organism disappears from the blood within 24 hours, and the patient becomes convalescent in a few days The value of highly skilled nursing is therefore much diminished The nurse should wear gloves while attending to the patient and wash her hands well in soap and water and afterwards rinse them in 1/2 000 perchloride of mercury Diet Patients undoubtedly do better when given an adequate dietary of 2 000 to 3 000 calories which prevents tissue waste The patient should drink enough fluid to prevent water depletion A check should also be kept on the electrolyte balance (see Chapter VI) The following diet can be given with a value of about 2 300 calories

| | |
|---|---|
| 7 a m | Milk 4 oz , water 2 oz cream ½ oz lactose ½ oz. 1 rusk and ¼ oz. butter |
| 9 a m | Beef tea well salted 1 cup |
| 11 a m | Milk 4 oz water 2 oz cream ½ oz lactose ½ oz |
| 1 p m | Mashed potato with butter 2 tablespoonfuls |
| 3 p m | Lightly boiled or poached egg with rusk or toast and butter (¼ oz ) |
| 5 p m | Cornflour or well boiled rice or egg custard 5 oz and cream 1½ oz. |
| 7 p m | Beef tea 1 cup |
| 10 p m | Milk 4 oz water 2 oz cream ½ oz. lactose ½ oz 1 rusk or toast and ¼ oz butter |

Two night feeds of milk 4 oz water 2 oz cream ½ oz. lactose ½ oz The milk feeds can be flavoured with tea coffee cocoa or chocolate Further additions can be made to the diet when the morning tempera ture falls to normal The mouth should be cleansed after each feed with a swab dipped in glycerin and lemon The bowels The stools should be examined daily for undigested food or blood or membrane If there is constipation an enema may be given on alternate days or liq magnes bicarb fl oz 1 by mouth If there is diarrhœa meat extracts and cream should be omitted and the milk should be peptonised If the diarrhœa still persists a starch and opium enema (starch gr 60 water fl oz 2 and tnc opii m 30) should be administered Meteorism is likely to occur if too much milk is given For its relief turpentine stupes can be applied to the abdomen the flannels being wrung out in water containing ol terebinthin m 60 in 2 pints of water If the temperature rises above 104 F it should be lowered 2 degrees by tepid sponging Chloramphenicol should be given by mouth in doses of 50 mg /lb body weight For an adult the dose is 1 G every 8 hours for 3 or 4 days until the temperature falls to normal subsequently mg 500 are given t i d for 2 to 3 weeks Despite the normal temperature the risk of hæmorrhage and perforation remains during the third week. Aureomycin and terramycin are also effective

Complications should be treated as follows

Hæmorrhage No food should be taken for 24 hours An injection of

above, together with the recrudescences and relapses which ensue at times The most important complications are —Intestinal (*a*) Hæmorrhage Especially during the third week The temperature falls the *pulse becomes rapid and feeble there is pallor, and air hunger may* ensue Blood is seen in the motions in varying amounts (*b*) Perforation and peritonitis These are likely to occur at the end of the third week, especially in the ambulant patient There is abdominal pain which may be referred to the tip of the penis, owing to the contiguity of the bladder The patient s expression becomes drawn and anxious The *effect on the temperature varies, it may or may not fall The pulse is* more frequent There is some limitation of abdominal movement with respiration the abdomen may be a little more swollen tenderness and rigidity may be present and the patient usually lies with the legs drawn up Venous thrombosis The left femoral vein is usually affected often in the fourth week Respiratory (*a*) Laryngitis or ulceration of laryngeal cartilages (*b*) Bronchitis (*c*) *Pneumonia in the third week* or later or at the onset (pneumo typhoid) (*d*) Pleural effusions Cardiac (*a*) Myocardial degeneration (*b*) Endocarditis and pericarditis are rare Neuritis especially causing the tender toes

Other complications include Otitis and parotitis which are not rare, meningitis cerebral thrombosis or embolus, myelitis nephritis typhoid spine (thickening of the intervertebral discs, it usually occurs late in the disease and the pain is very severe) periostitis, boils acute cholecystitis infarction of the spleen and suppuration of the mesenteric glands White leg following venous thrombosis or gall stones may result as sequelæ of typhoid fever

**Prognosis** This has been much improved by the use of chloramphenicol and the mortality rate has probably fallen to about 1%

**Treatment** *Prophylactic* The disease is preventable Contacts and all exposed to infection should be inoculated inoculation does not increase the risk of infection A mixed vaccine (T A B) containing 1 000 millions typhoid and 500 millions paratyphoid, A and B in 1 ml is used 0 5 ml is first given and after 10 days 1 ml is injected subcutaneously This vaccine may be combined with 1 ml of tetanus toxoid in each dose and repeated at an interval of 1 month Pulmonary tuberculosis is a contra indication The immunity conferred lasts about 2 years In an epidemic water and milk should be boiled and carriers sought for There is no medical treatment which will inevitably cure a chronic carrier In some cases cholecystectomy has been successful When cholecystectomy has failed the carrier state has been abolished in a few cases by giving 200 000 units of penicillin every 2 hours for 6 to 8 days, together with sulphathiazole 1 G by mouth every 3 hours Chloramphenicol does not appear to affect the carrier state All cases must be notified *Isolation period* The patient must be isolated for about 5 to 6 weeks and on discharge the fæces and urine should be free from typhoid bacilli on two successive examinations at an interval of 1 week. Should the patient continue to excrete typhoid bacilli after 3 months he becomes a carrier A positive Vi agglutination does not indicate that the individual is a carrier

Br suis of swine In Great Britain only bovines are infected in America swine are also infected Both Brucella groups may cause undulant fever in man Fifteen to twenty per cent of adult bovines in Great Britain were infected before the late war but the number has now been reduced by vaccination Cream is a less common source of infection and butter and cheese are doubtful sources Veterinary surgeons and farmers may be infected through the skin while looking after parturient cows Malta fever is usually contracted through drinking goats milk Water may be contaminated from goats and the disease may also be transmitted through cheese The disease occurs in almost every part of the world *Predisposing causes* Sex Males predominate possibly because of an occupational incidence Age 15 to 45

**Pathology** The brucella lodges in the cells of the reticulo endothelial system especially in the lymph glands liver spleen and bone marrow Small granulomata are formed Caseation does not occur but there may be suppuration The testis ovary or meninges may be affected

**Incubation Period** 5 to 30 days usually 2 to 4 weeks

**Clinical Findings** The patient is gradually taken ill with malaise headache and pain in the back and limbs The onset may be sudden with a rigor Pains may also be felt in the jaws eyeballs or testicles In some cases the throat is sore and swallowing is painful

*On Examination* The most striking feature is the continued fever The temperature usually rises irregularly and remains remittent for 1 to 4 weeks After falling to normal the temperature may rise again in one or two days but the second wave is generally shorter than the first The temperature may then settle to normal with an occasional rise to 99 F or for a prolonged period such as 8 months irregular rises may occur The pulse is usually slow about 80 There is often marked sweating in the early morning and transient painful swellings may occur in the joints The bowels tend to be constipated The spleen is palpable in some cases. Less frequently the liver and lymph glands may be enlarged Epistaxis may occur late in the disease A vesicular eruption may be seen on the palate The blood There is leucopenia with a relative lymphocytosis of about 50% and some anæmia The agglutination test is usually first positive between the sixth and fourteenth days An agglutination in a dilution of 1/80 is considered to be diagnostic Often agglutination in a dilution of 1/1 000 or more is obtained The blood culture is often positive but the organism may not grow for 19 days or longer under anaerobic con ditions *Varieties* Mild attacks probably frequently pass unnoticed A severe septicæmic form is also described

**Differential Diagnosis** The disease must be distinguished from other causes of prolonged fever sweating and joint pains such as enterica infections tuberculosis malaria septic infections rheumatic fever and influenza The diagnosis rests upon the agglutination test, the leucopenia and the isolation of the organism from the blood There is also an intradermal test performed by injecting 0 1 ml of brucellin With a positive reaction an œdematous area with surrounding erythema 2 to 6 cm in diameter appears in 4 to 48 hours This is an indication

*morphin sulph gr ¼ should be given If the bleeding persists a starch* and opium enema should be administered If the patient is very exsanguinated he should receive a drip blood transfusion An enema should be given if the bowels have not moved in 4 days after a preliminary rectal injection of 4 oz of warm olive oil

Perforation A laparotomy should be performed immediately, preferably under a spinal or local anæsthetic

General instructions The urine should be mixed with an equal volume of 1/20 carbolic acid and allowed to stand covered over for 2 hours before being thrown into the drain The fæces cannot be adequately sterilised by clinical disinfectants but should be mixed with an equal quantity of 1/20 carbolic acid for 2 hours Where the water carriage system is not perfect they should be mixed with paraffin and sawdust and burnt Sheets should be changed directly they are soiled with excreta and put into a bucket containing 1 in 10 carbolic acid for 6 hours Fly papers should be hung in the sick room

## The Paratyphoid Fevers

Paratyphoid fevers bear a close resemblance clinically and bacteriologically to typhoid fever, they are caused by the Bacterium paratyphosum A B or C (S paratyphosa, S schottmulleri or S hirschfeldii)

These organisms can be identified by cultural and agglutination reactions Para A is rare in England, but Para B occurs fairly often Para C is met with chiefly in the Eastern Mediterranean and Mesopotamia The diseases are spread in the same way as typhoid by contact carriers and infection of food but contaminated water does not appear to be so frequently incriminated Chinese frozen whole eggs appeared to be the source of two outbreaks of Para B epidemics in England in 1955 Paratyphoid fever as opposed to typhoid fever can also be transmitted from animals to man

Clinically paratyphoid fevers may begin comparatively suddenly with shivering and pyrexia but the diseases are usually less severe and of shorter duration than typhoid fever, and ulceration of the intestines is less liable to occur The colon is ulcerated in some cases and the causative organism is more likely to be found in the fæces than it is in typhoid fever The temperature curve is more irregular A *respiratory* type of paratyphoid is described with bronchitis or pneumonia in which paratyphoid bacilli are present in the sputum and a septicæmic type with high remittent fever pneumonia arthritis, meningitis, pyelonephritis endocarditis cholecystitis or pelvic abscesses The treatment is identical with that of typhoid fever (see p 582)

## Brucellosis

(*Undulant Fever Malta Fever Abortus Fever*)

**Definition** A disease due to a specific micro organism and conveyed by milk

**Etiology** The Brucella group of organisms includes the Br melitensis infecting goats and sheep the Br abortus of bovines and the

and Tabardillo in Mexico The reservoir of the infection is in mice and rats

**Pathology** Typhus nodules are found microscopically post mortem in the skin brain and other organs They are due to localised necrosis and dilatation of small vessels The spleen is enlarged and soft and petechiæ may be seen in various mucous membranes

**Incubation Period** 5 to 21 days usually 12 to 14 days

**Clinical Findings** There may be a prodromal period with malaise for a few days The patient is usually suddenly taken ill with frontal or occipital headache pains in the back and limbs shivering nausea or rarely vomiting

*On Examination* The patient is flushed and looks rather drowsy the eyes are congested and the tongue coated The temperature is raised to about 100 F The course of this disease has been considerably modified by immunisation and by the use of chloramphenicol and aureomycin The following description is that of an unimmunised case not treated by these antibiotics

The rash appears on the fourth or fifth day first as a subcuticular mottling ' the spots being under the skin and later papules develop These are rose pink fading on pressure and resemble typhoid spots They enlarge become darker brownish and do not fade on pressure Small petechiæ may be seen between the spots The whole rash is called a mulberry rash It appears first on the trunk and spreads to the limbs the face is usually exempt but is red and swollen The rash fades during the second week and leaves some staining It does not appear in crops The skin emits a peculiar mousy odour The temperature This usually rises irregularly during the first 4 days to about 103° F there is then a continued fever for about a week when the crisis occurs about the tenth or fourteenth day The fall is critical but may take 2 to 3 days before normal is reached The pulse increases in rate with the rise in temperature The respirations are usually increased to about 20 per minute The blood There is a leucocytosis with excess of monocytes (up to 15%) A special agglutination is given by the serum called the Weil Felix reaction The serum agglutinates B proteus strain OX19 It is often found that by the end of the first week the titre reaches 1/200 and by the end of the second week 1/1 000 or more A rising titre is characteristic of an active infection The slide test is often positive by the end of the first week A drop of blood is mixed with a loopful of methylene blue coloured proteus OX19 suspension on a slide Agglutination is shown by the formation of blue aggregates visible to the naked eye The cerebro spinal fluid is clear but may contain an excess of globulin and leucocytes

The general condition throughout the febrile period is one of extreme prostration an excited delirium may occur during the first week followed by coma vigil in the second week in which the patient lies semi conscious with eyes partially open and muscles twitching The patient may be tortured by fearful delusions The pupils are usually small and the knee jerks are absent the tongue becomes dry black and cracked The bowels are often constipated early and diarrhœa

that the patient has had a brucella infection in the past, or has one at the time of the test

**Course and Complications** As described above the course is characterised by a tendency to relapse and so it is difficult to say when the disease is cured In some cases the disease lasts only a few days in others for many years Complications include bronchitis orchitis abortion, mastitis parotitis, arthritis and pains in the extremities

**Prognosis** The mortality rate is 2 to 3%, and the disease is often serious owing to the tendency to prolonged invalidism

**Treatment** *Prophylactic* Only pasteurised or boiled milk should be drunk The treatment of infected cows is still undecided Living non virulent vaccines are usually administered to check the abortion, but there is a risk that the cows will then become chronic carriers eliminating the organisms in their milk

*Curative* The patient should be put to bed and a mild aperient given The diet should be liquid during the pyrexial waves, such as boiled or pasteurised milk dextrose orangeade barley water and meat extracts but nourishment should be increased during the apyrexial intervals Pains may be relieved by aspirin gr 10 t d s or phenacetin gr 5 as required If the temperature is above 103° F it should be lowered two degrees by tepid sponging The most favourable results have been obtained with aureomycin in doses of 30 to 50 mg /kg body weight daily For an adult 0 5 G may be given every 6 hours for 2 to 3 weeks A second course should be given if relapse occurs It is probable that equally good results are obtainable with terramycin and Achromycin In the more severe cases especially those infected with Br melitensis or Br suis intramuscular injections of streptomycin 1 G should be given every 12 hours for a week followed by 0 5 G every 12 hours for 2 weeks The urine and fæces should be disinfected as for typhoid fever

## Typhus Fever

### (*Spotted Fever*)

**Definition.** An acute infectious disease characterised by fever a typical rash profound toxæmia and nervous symptoms

**Etiology** Typhus fever is caused by Rickettsia prowazeki oval filter passing organisms These are found in the patient (internal organs) and in lice (pediculi corporis and also capitis) which transmit the disease Typhus fever is spread by lice either by their bites or by their excreta being scratched into the skin The louse does not become infective for 6 to 7 days after biting a patient with typhus Flea borne and tick borne forms of typhus also occur Scrub typhus is described on p 737 *Predisposing causes* 1 Under nutrition and famine 2 Dirt and over crowding 3 Season Winter and spring 4 Age and sex All ages and both sexes are affected 5 Locality It is more common in Eastern than in Western Europe It occurs in North and Central Asia North Africa and near the Andes in South America It is unknown in the tropics except at great altitudes It occurs endemically and in epidemics A mild form (flea borne) is called Brill s disease in America

hour for 3 doses followed by 0 5 to 1 5 G every 6 hours The drug should be continued for 18 hours after the temperature is normal

## Q Fever

**Definition** An acute febrile illness often accompanied by pneumonitis due to a Rickettsial infection The name Q fever was originally chosen by Derrick who first described it in 1937 in Queensland until a fuller knowledge should allow a better name It has subsequently been recognised in Europe Africa and America

**Etiology** The causative organism is the Rickettsia burneti Human beings are infected by occupational exposure to dairy cattle, and by drinking raw milk from infected cows The R burneti has been found in the bandicoot a small Australian bush animal and in various ticks

**Pathology** Rickettsiæ may be isolated from the lungs spleen testes brain and kidneys The microscopical appearance of the lungs resembles those found in psittacosis and virus pneumonias

**Incubation Period** 12 to 15 days

**Clinical Findings** The disease has a sudden onset the temperature rising to 103 or 104 F It may fall to normal once or twice in the 24 hours The pulse is slow and in severe cases there are rigors and heavy sweats Prostration delirium and coma have been described There is severe occipital and retro orbital headache and generalised muscular aching In a few days a dry irritating cough appears and a little blood stained sputum may be expectorated

*On Examination* Small areas of high pitched bronchial breathing with fine rales may be heard in the lungs Occasionally there is a lobar consolidation pleural friction or pleural exudation The spleen may be palpable There is no rash as occurs with other Rickettsial infections X ray examination of the lungs shows patchy rather uniform shadows which may persist after the patient has apparently recovered The temperature often falls to normal after a week but it may persist for 3 to 4 weeks There is usually no anæmia and the white cell count is often normal although there may be a relative or absolute lymphocytosis The Rickettsiæ may be found in the blood less often in the urine The serum taken on the twelfth and twentieth days usually shows an increase in agglutination titre of from 1 in 4 to 1 in 20 with rickettsial emulsions

**Differential Diagnosis** Most cases in this country are probably mistaken for virus pneumonia Derrick considered the possibility of typhus fever undulant fever typhoid and paratyphoid fever leptospirosis and influenza Diagnosis is established by the agglutination test which may rise to 1 in 80 and by finding the causative organism in the blood or urine In epidemics the source may be traced to the milk supplied from a special farm although the infected cows appear healthy

**Prognosis** The death rate is about 1 in 500

**Treatment** Aureomycin should be given in doses similar to those for typhus fever (see p 588) The response is often rapid and dramatic

may occur in the second week thirst is marked and there may be some deafness, the spleen is usually just palpable In a favourable case, when the temperature falls the condition rapidly improves

*Varieties* 1 *Mild typhus* 2 *Severe or blasting typhus* (*Typhus siderans*), which is rapidly fatal 3 *Typhus sine eruptione* may occur, in which the Weil Felix test is positive

**Differential Diagnosis** Typhus is very rare in England, and when an isolated case occurs it is most likely to be mistaken for typhoid fever or measles Other conditions which may require exclusion are Influenza, cerebro spinal fever, malignant malaria encephalitis purpura, relapsing fever or uræmia The marked prostration, typical rash and Weil Felix reaction serve to differentiate typhus

**Course and Complications** A relapse is extremely rare but a second attack may occur Complications include Laryngitis bronchitis, myocardial degeneration venous thrombosis, otitis media and nephritis Permanent mental deterioration inability to concentrate or headaches may be sequelæ

**Prognosis** Typhus fever is a serious disease and the mortality increases with the age of the patient The outlook has however, been much improved by the use of antibiotics

**Treatment** *Prophylactic* Good protection is obtained by the vaccine prepared by growing the Rickettsia prowazeki in the yolk sac of chicken embryos Three doses of 1 ml are given at weekly intervals Freedom from lice is all important Native populations should be deloused by D D T (dichlor diphenyl trichlorethane) powder during epidemics *Quarantine period* Contacts should be deloused and quarantined for 15 days *Isolation period* The patient should be isolated for 5 weeks

*Curative* The patient should be taken to an isolation hospital his clothes removed and deloused by D D T powder the axillary and pubic hair shaved the head closely clipped and treated with sassafras lotion (ol sassafras (B P C ) fl oz ¾ ol amygdal m 60) with a stiff brush to destroy nits and bathed in soap and water The attendants should have their hair and persons protected from lice by caps overalls and D D T powder and they should wear rubber gloves The patient is put to bed in an airy room Plenty of fluids should be given by mouth to aid toxin elimination The skin should be tepid sponged twice or thrice a day The mouth should be cleaned after feeds with glycerin and borax or glycerin thymol co (B P C ) The bowels should be opened as required with an enema if there is retention of urine catheterisation at eight hourly intervals must be performed For the excited delirium sod phenobarbiton gr 1 and sod brom gr 30, or paraldehyde m 60 to 120 may be given by mouth or it may be necessary to inject subcutaneously hyoscin hydrobrom gr 1/200 If there are signs of collapse cardiac stimulants such as hot drinks whisky fl oz 2 in 24 hours or the subcutaneous injection of Coramine 1 5 ml may be given four hourly With the use of terramycin or aureomycin the disease is arrested and the temperature usually falls to normal in about 48 hours For an adult the initial dose is 1 G every

hour for 3 doses followed by 0 5 to 1 5 G every 6 hours The drug should be continued for 48 hours after the temperature is normal

## Q Fever

**Definition** An acute febrile illness often accompanied by pneumonitis due to a Rickettsial infection The name Q fever was originally chosen by Derrick who first described it in 1937 in Queensland *until a fuller knowledge should allow a better name* It has subsequently been recognised in Europe Africa and America

**Etiology** The causative organism is the Rickettsia burneti Human beings are infected by occupational exposure to dairy cattle and by drinking raw milk from infected cows The R burneti has been found in the bandicoot a small Australian bush animal and in various ticks

**Pathology** Rickettsiæ may be isolated from the lungs spleen testes brain and kidneys The microscopical appearance of the lungs resembles those found in psittacosis and virus pneumonias

**Incubation Period** 12 to 15 days

**Clinical Findings** The disease has a sudden onset the temperature rising to 103 or 104° F It may fall to normal once or twice in the 24 hours The pulse is slow and in severe cases there are rigors and heavy sweats Prostration, delirium and coma have been described There is severe occipital and retro-orbital headache and generalised muscular aching In a few days a dry irritating cough appears and a little blood stained sputum may be expectorated

*On Examination* Small areas of high pitched bronchial breathing with fine rales may be heard in the lungs Occasionally there is a lobar consolidation pleural friction or pleural exudation The spleen may be palpable There is no rash as occurs with other Rickettsial infections X ray examination of the lungs shows patchy rather uniform shadows which may persist after the patient has apparently recovered The temperature often falls to normal after a week but it may persist for 3 to 4 weeks There is usually no anæmia and the white cell count is often normal although there may be a relative or absolute lymphocytosis The Rickettsiæ may be found in the blood less often in the urine The serum taken on the twelfth and twentieth days usually shows an increase in agglutination titre of from 1 in 4 to 1 in 20 with rickettsial emulsions

**Differential Diagnosis** Most cases in this country are probably mistaken for virus pneumonia Derrick considered the possibility of typhus fever undulant fever typhoid and paratyphoid fever leptospirosis and influenza Diagnosis is established by the agglutination test which may rise to 1 in 80 and by finding the causative organism in the blood or urine In epidemics the source may be traced to the milk supplied from a special farm although the infected cows appear healthy

**Prognosis** The death rate is about 1 in 500

**Treatment** Aureomycin should be given in doses similar to those for typhus fever (see p 588) The response is often rapid and dramatic

the temperature rapidly falling to normal, the headache disappearing and the patient feeling well and asking to get up

## Erysipelas

**Definition** An acute infective disease characterised by fever and a typical inflammation of the skin

**Etiology** Erysipelas is due to infection with different types of group A hæmolytic streptococci *Predisposing causes* 1 Alcoholism 2 General debility and defective sanitation 3 Season Autumn and winter 4 A previous attack Recurrences are very common 5 Age It occurs in new born infants and subsequently chiefly between the ages of 20 and 60

The organisms enter the skin through a minute abrasion or through operation wounds or birth injuries (puerperal erysipelas) They are conveyed by direct contact or by contaminated hands or instruments or the infection may occur apparently independently

**Pathology** The organisms are found chiefly in the lymphatics at the edges of the inflamed area of skin, and spread to the subcutaneous tissues may cause suppuration Toxæmia is often severe

**Incubation Period** 2 to 3 days or longer

**Clinical Findings** The patient, who is usually an adult, may give a history of some trivial injury to the face, or may be recovering from an operation or childbirth There is usually a sudden onset with shivering, headache malaise, and at times vomiting and a sore throat A burning sensation is then felt on a certain area of the skin

*On Examination* A bright red patch is seen with a definite raised margin This spreads and the skin becomes tense and shiny, and vesicles or blebs containing yellowish fluid may form on the red area This fluid usually does not contain streptococci

Facial erysipelas usually begins near the nose or inner canthus of the eye The face may become very bloated and the eyelids so œdematous that the patient cannot see, the ears also become red and tense and the tongue is very furred The tissues are at this stage very painful on pressure and the skin feels tightly stretched The glands in the neck are enlarged Headache insomnia and noisy delirium may be troublesome

Erysipelas may spread from one part of the skin to another (wandering erysipelas), or the lesion may first appear at such sites as the back around the umbilicus in newborn infants, around the vulva during the puerperium and around any operation wound which is usually a " clean " one The fauces may be red and swollen (faucial erysipelas) or the mucous membrane of the nose affected The temperature rises rapidly to 103 or 104° F but usually falls quickly with penicillin treatment The blood shows a polymorphonuclear leucocytosis The urine usually contains albumin When the rash fades it is followed by desquamation

**Differential Diagnosis** Erysipelas must be diagnosed from simple inflammations In the latter there is not a raised, red edge, and the central part of the inflamed area is more angry looking than the periphery The constitutional disturbance is not as great

**Course and Complications** Relapse may develop after the temperature has been normal for a day or so or several months later Complications include septicæmia subcutaneous abscesses and bronchopneumonia

**Prognosis** Erysipelas is frequently mild but the outlook is more serious in an alcoholic, in the new born and in the aged

**Treatment** The patient should be kept in bed and isolated until the temperature is normal and the lesions healed The diet should be liquid or semi solid while the temperature is raised Plenty of fluids should be drunk and the bowels opened daily with salines or enemata Paraldehyde fl oz ½ to ¾ may be given to an adult for insomnia Noisy delirium may be checked with an injection of hyoscin hydrobrom gr 1/200 to 1/100 For headache an ice bag may be applied Brandy or whisky fl oz ½ six hourly should be given to alcoholics

*Local Treatment* The affected skin may be covered with lint soaked in a cold saturated solution of magnesium sulphate The eyes should be washed out with boric lotion and a drop of Albucid 10% instilled every 4 hours if they become inflamed

300 000 units of a procaine penicillin may be injected intramuscularly every 12 hours for 5 to 7 days This is the treatment of choice

## CHAPTER IX

# INFECTIOUS DISEASES OF KNOWN AND DOUBTFUL ETIOLOGY

### Syphilis

*Definition* A venereal and general disease caused by a specific protozoon

Etiology The causative agent is the Treponema pallidum (Spirochæte pallida) Infection is usually by direct transmission in sexual intercourse The primary sore and secondary lesions, such as condylomata, are very infectious and the treponemes may also be present in the saliva and urine in the secondary stage Localised gummata such as may occur in the throat are also sources of infection Syphilis less often results from other causes as by contact with an infected article such as a cup or a pipe or the primary lesion may occur on a doctor's examining finger (syphilis innocens) The disease may be transmitted by an infected mother to her offspring (congenital syphilis) The organisms usually enter through an abrasion in a mucous membrane

Pathology The initial lesion is the primary sore or chancre The treponemes pass to the neighbouring lymphatic glands and rapid dissemination by the blood stream ensues probably as soon as, or even before the chancre is visible The primary sore is usually genital but extragenital chancres may occur on the lip, tonsil, tongue breast finger and elsewhere The secondary stage is the clinical *manifestation* of generalisation of infection , lymphatic glands are enlarged especially the posterior cervical axillary and epitrochlear Various rashes occur due to reactions around cutaneous blood vessels The kidneys may show evidence of nephrosis Gumma formation is the characteristic feature of the tertiary stage the gumma being a granuloma which is comparatively avascular It may occur in the skin the heart or lungs the liver, the central nervous system bones and in other tissues *The* arteries are especially affected in syphilis there being early a round celled infiltration of the adventitia The inflammatory change *spreads* inwards along the vasa vasorum to the media, where rupture of elastic fibres occurs In the aorta this predisposes to aneurysm The mouths of the coronary arteries are constricted but the vessels themselves are unaffected Endarteritis obliterans may occur in the smaller vessels Treponemes are most numerous in the primary sore and the neighbouring glands they occur in the blood cerebro spinal fluid and in the cutaneous lesions they are most scanty in the tertiary lesions but lie dormant there being sheltered from blood borne spirochætocidal drugs owing to the interferences with the blood supply

## Congenital Syphilis

**Clinical Findings** *A history is often obtained that the mother* has had a series of miscarriages or still births before a living child is born At birth the infant may be normal in appearance, or small The skin may be sallow (*café au lait*) the face old looking and the cry rather squeaking A bullous or pustular eruption may be present at birth The various manifestations of congenital syphilis are best classified under the age periods at which they are likely to appear

*At Birth* Much hair on the head (syphilitic mop) Aged appearance Bullous rash (syphilitic pemphigus) especially on the palms and soles White pneumonia (due to fibrosis of the lungs)

*Three to Four Weeks* Syphilitic roseola especially around the buttocks snuffles' due to rhinitis otitis media Choroiditis and iritis Paroxysmal hæmoglobinuria

*Three to Four Months* Epiphysitis causing apparent paralysis of limbs (pseudo-paresis) Rhagades (fissures) at the angles of the mouth which leave radiating scars on healing Condylomata in the perineum or under the arms Enlargement of the spleen and liver Gumma of the testicle

*Six to Twelve Months* Iritis Bossing of the skull on the frontal and parietal bones (Parrot s nodes or hot cross bun appearance) and cranio tabes (softened areas of bone in the skull) are probably rickety changes

*Second Year* Dactylitis the phalanges of the fingers or toes being swollen Depression of the bridge of the nose (saddle bridge) Hydro cephalus and idiocy

*Childhood and Later* Keratitis especially between 6 and 12 years The teeth The central permanent upper incisors are notched at the cutting edge which is narrower than the base and the teeth are spaced (Hutchinson s teeth) The first permanent molars may be dome shaped owing to failure of development of the central portion of the crown (Moon s teeth) Deafness may occur suddenly from gummatous destruction of the internal ear Periostitis The tibiæ become curved forwards and thickened (sabre tibia) Painless swelling of joints such as the knee (hydrarthrosis) Nervous lesions may be manifest such as dementia juvenile tabes or general paralysis Diabetes insipidus may result from a basal syphilitic meningitis The blood Wassermann reaction is usually positive up to puberty but may become spon taneously negative later in life

The cerebro spinal fluid This shows changes characteristic of syphilis (see p 410) in about 40% of cases depending upon whether the central nervous system is involved

**Differential Diagnosis** Hutchinson s diagnostic triad is of value in childhood the stigmata are interstitial keratitis the typical incisor teeth and deafness In infants a napkin rash must not be mistaken for a syphilitic roseola The history appearances of the child and positive Wassermann reaction of the child and its mother are usually diagnostic

**Course and Complications** The victim of congenital syphilis may be very slightly or very severely affected Fresh manifestations are

liable to show themselves *in late childhood or early adolescence* as mentioned above. Usually the child grows up, but is often stunted, and the intelligence may be below normal. Secondary infections are liable to occur.

**Prognosis** This depends upon the intensity of the infection and the organs affected. The virulence usually diminishes in proportion to the length of *time the mother* has been *infected*.

**Treatment** *Prophylactic* The mother should be *given a course of* anti syphilitic treatment before or during pregnancy. This consists of the intramuscular injection of 600 000 units of a procaine penicillin daily for 14 days. In every subsequent pregnancy the procaine penicillin is repeated for 14 days.

*Curative* The child should be treated directly the disease is recognised. Congenital syphilis in an *infant or child* is treated by the daily intramuscular injection of 300 000 units of a procaine penicillin for 14 days. Four such courses are given during a period of 2 years. Interstitial keratitis should be treated by an ophthalmologist.

### Acquired Syphilis

**Incubation Period** This *is usually about 28 days probably never* less than 10 days but it may be prolonged to 12 or 13 weeks.

**Clinical Findings** *Primary Stage* The patient gives a history that about 4 weeks after sexual intercourse he noticed a small red spot on the penis. This enlarged but was painless and gradually formed a sore.

*On Examination* The sore has an indurated cartilaginous feeling (hard or Hunterian chancre) after 2 to 3 weeks. Serum exudes from it on slight scarification and this serum is teeming with treponemes. The chancre may be within the meatus of the urethra, or at other sites *as mentioned on p 592. If untreated the chancre heals in about 6 to 8 weeks leaving a scar. The neighbouring lymphatic glands enlarge,* and treponemes are found in the fluid obtained by gland puncture. The glands may suppurate if there is a mixed infection, constituting a bubo. In about another fortnight enlargement of glands is also noted at the back of the neck under the arms and in the epitrochlear regions. The blood Wassermann reaction becomes *positive* about *8 weeks after infection* and changes *in the* protein and cell contents of the cerebrospinal fluid are usually present.

*The Secondary Stage* This is noted about 4 weeks or later (up to 6 months) after the appearance of the primary sore and is characterised by systemic disturbance as shown *by headache sore throat* anaemia, *skin rashes generalised* glandular enlargement and at times by nephrosis. The patient may also complain of pains in the limbs due to synovitis or periostitis. Various rashes may occur; the syphilitic roseola is ham coloured or brownish and is seen on the trunk and extremities. Brownish pigmentation with areas of *leucoderma may develop* on the neck, *especially in dark* haired patients. Papulo pustular or scaly eruptions *may* also appear on the body and limbs; the epithelium may become heaped up in a conical mass *forming* rupia. Soft warty excrescences or

condylomata are apt to form on moist surfaces which are not kept clean, as around the anal margin vulva and under the breasts The throat may be very red and "snail track" greyish white streaks may be seen on the fauces or soft palate and ulceration of the tonsils White mucous patches may occur inside the cheeks Alopecia of a general or patchy type is liable to occur Chronic laryngitis may be present. Jaundice may develop with an enlarged liver or in a severe form with acute yellow atrophy The blood There is anæmia treponemes may be present and the Wassermann reaction is positive The cerebro spinal fluid The Wassermann reaction is often positive and other changes such as excess of globulin, increase of lymphocytes and a characteristic colloidal gold curve (Lange s test) may be present (see Fig 24)

*Tertiary Stage* This stage may emerge directly from the preceding one, or be separated from it by an interval of many years It is characterised by the formation of gummata The gummata may appear in various parts of the body In the skin and subcutaneous tissues as ulcerating nodules which separate leaving punched out ulcers and on healing form thin paper like scars in muscles forming painless swell ings which eventually ulcerate and in bones causing severe pains usually intensified at night Gummata may also form in internal organs such as the liver the testis lungs pituitary and the adrenal The heart may be involved the gumma giving rise to disturbances of conduction according to its situation or to localised myocardial degeneration Syphilitic arterial changes give rise to cardio vascular or renal degenera tion The various neurological diseases such as general paralysis tabes dorsalis cerebral gumma meningitis and meningo myelitis are described in the chapter on nervous diseases The alimentary tract may be affected Glossitis leukoplakia localised gumma or ulceration may occur in the tongue There may be ulceration of the tonsils or gumma formation in the œsophagus less frequently in the stomach Iritis and otitis may also occur as tertiary manifestations A prolonged continued temperature lasting for several weeks may occur at times during the tertiary stage The various results produced by tertiary syphilis are more fully considered in the chapters dealing with the heart lungs alimentary tract etc

**Differential Diagnosis** In the primary stage the diagnosis should be established by finding treponemes in the serum from the chancre and treatment should not be delayed until the blood Wassermann reaction becomes positive Chancroid herpetic and scabietic lesions may cause confusion In the later stages the diagnosis is usually established by the Wassermann reaction of the blood and the cerebro spinal fluid Combined lesions may occur such as an epithelioma of the tongue in a patient who is also a victim of syphilis A positive Wassermann reaction may also be found during the pyrexial stage of malaria although the patient is not suffering from syphilis and it has been recorded in other diseases such as scarlet fever typhus fever yaws etc

*Course and Complications* The disease usually pursues a very chronic course and the treponemes are apt to lie dormant in various

parts of the body, shut off from the blood stream Later they give rise to severe reactions in the form of tertiary lesions

**Prognosis** Syphilis is a very serious disease and it is impossible to say in any individual case what course it will pursue A mild initial lesion may be followed years later by crippling cardiac complications or fatal nervous affections Efficient treatment in the earliest stages holds out the best hope of a permanent cure

**Treatment** *Prophylactic* Avoidance of irregular sexual intercourse is the best preventive Apart from this, protection is afforded by the use of a condom or sheath during intercourse and the immediate washing of the genitalia in soap and water after intercourse The patient should not marry for 2 years from the beginning of the treatment, and his Wassermann reaction should be negative

*Curative* As soon as the chancre appears and the diagnosis is established by finding treponemes in the smear a course of treatment should be instituted The patient must not take any alcohol and if feverish he should be in bed Procaine benzylpenicillin in oil containing 2% aluminium monostearate should be injected intramuscularly daily in doses of 600 000 units for 14 days During the first year the blood Wassermann reaction is determined every 3 months and every 6 months during the second and third years The cerebro spinal fluid is examined at the end of the first and third years If there is a relapse the treatment is repeated

For late syphilis including the latent form and the tertiary manifestations, 600 000 units of a procaine penicillin are injected intramuscularly daily for 4 weeks After 6 months the penicillin course is repeated Further courses are given during the next 2 years if the serological and clinical results are not satisfactory

*The Jarisch Herxheimer Reaction* This may result from penicillin treatment It may occur on the day after the first or second injection It is probably due to an increased activity around the site of the lesions, caused by liberation of toxins from the treponemes destroyed Thus the rash may be more marked and the Wassermann reaction become more strongly positive If the lesions are cardiac laryngeal or cerebral very grave results may ensue from vascular changes in the affected areas such as heart failure laryngeal obstruction or paralysis To prevent these reactions the initial dose of penicillin should be small (see p 407)

## Gonorrhœa

**Definition** A veneral disease caused by infection with a specific micrococcus

**Etiology** The cause is the Neisseria gonorrhœæ (gonococcus) Infection in man is usually by sexual intercourse Infants may have their eyes infected at birth if the mother is suffering from the disease Gonorrhœal vaginitis of children may become epidemic in institutions due to infected towels or clothes

**Pathology** The gonococcus penetrates mucous membranes such as line the urethra or vagina It may spread directly to the uterus,

Fallopian tubes peritoneum glands of Bartholin prostate seminal vesicles bladder pelvis of the kidney or rectum It may be carried by the blood stream to the heart meninges joints muscles tendons or eyes The toxins may cause such lesions as peripheral neuritis, kerato derma or iritis Infection may be conveyed by the finger to the eyes, causing ophthalmia

**Incubation Period.** This is usually from 3 to 10 days

**Clinical Findings** Acute gonorrhœa is characterised by acute urethritis and in the female also by cervicitis and vaginitis The secondary complications which were formerly so common are likely not to occur with early penicillin treatment They included Acute arthritis meningitis or myelitis pericarditis endocarditis and myocard itis peripheral neuritis and septicæmia

*Gonorrhœal Arthritis* The patient is usually a young adult of either sex who may be suffering from an attack of acute gonorrhœa or who may have been infected previously He is suddenly taken ill with malaise and pain in one or more joints Often one joint only is affected such as the knee ankle wrist shoulder or elbow or the pain may be localised to the sterno clavicular, temporo mandibular spinal or sacro iliac joints

*On Examination* The affected joint if one of the large ones, is swollen tender and the skin over it may be slightly red and hot effusion is present in the joint and movement is painful There is also constitutional disturbance for the tongue is furred and the temperature and pulse rate are raised If fluid is aspirated from the joint it is sero fibrinous and may contain gonococci In women there is usually a vaginal discharge and in men a urethral discharge may be present before or after prostatic massage A swab taken from the cervix uteri or from the urethra after prostatic massage often shows gonococci

*Varieties* Chronic hydrarthrosis which is painless and insidious in onset may occur or subacute polyarthritis

**Differential Diagnosis** The commonest error is to diagnose the *condition as acute rheumatism or some other variety of acute infective* arthritis The characteristic features are the history of gonorrhœa which however is often not forthcoming the special joints affected the presence of gonococci in the genito urinary discharge a positive complement fixation test and the response to specific treatment Salicylates fail to give relief

**Course and Complications** The joints usually recover with proper treatment, but stiffness may persist for long periods A chronic painless arthritis with effusion may ensue in some cases

**Prognosis** This is generally good

**Treatment** If the case is seen early a single injection of 600 000 units of a procaine penicillin usually effects a cure If gleet persists after adequate penicillin treatment it is due to non specific urethritis and should be treated with aureomycin For gonorrheal arthritis 600 000 units of penicillin should be injected every 12 hours for 5 to 10 days For vulvovaginitis and for secondary complications the penicillin

should be followed by a course of sulphathiazole Suppurative peri adenitis of Bartholin's glands requires aspiration of the pus and subsequent injection of 100 000 units of a water solution of penicillin The dose of sulphathiazole for an adult is 4 G followed by 2 G 6 hours later Subsequently, 1 G is given every 6 hours until a total amount of 30 G has been taken The urine should be kept alkaline by a potassium citrate mixture, 5 to 6 pints of fluid being taken every 24 hours In all cases the blood Wassermann reaction should be determined at the end of 2 and 6 months

## Rheumatic Fever

*(Acute Rheumatism)*

**Definition** An acute disease characterised by fever, joint pains and a liability to carditis

**Etiology** Rheumatic fever is probably due to infection through the throat and possibly through the intestine with group A hæmolytic streptococci the organisms passing by the blood stream to the sites of election the heart the joints subcutaneous tissues and meninges Some authorities believe that streptococci lodged in sites such as the tonsils produce a chronic allergic state which on stimulation results in acute rheumatism *Predisposing causes* 1 Familial diathesis The disease runs in families presumably due to deficient resistance and the case incidence points to contact infection 2 Age and sex Children and young adults are chiefly affected, females preponderating somewhat in childhood and males later 3 Social factors Over crowding poor food (possibly lacking in vitamin C) and damp houses are of importance 4 Climate and season Rheumatic fever prevails chiefly in temperate climates in England especially in the autumn and spring and epidemic years occur Statistics tend to show that the incidence of rheumatism in children corresponds closely with that of rainfall 5 Previous attacks These predispose to subsequent ones an epidemic of tonsillitis may precede one of acute rheumatism

**Pathology** It is not definitely known if the causative organisms circulate in the blood and settle in the joints and heart or whether the lesions are due to a toxæmia, the organisms present from time to time in the blood and tissues being secondary infective agents Post-mortem The heart Pericarditis dry or with effusion may be present Aschoff s sub miliary nodules may be found microscopically in the myocardium They are spindle shaped and contain connective tissue cells and fibroblasts Small sessile vegetations may be seen on the valves such as the mitral or aortic valve Subcutaneous nodules felt before death may not be found at autopsy these are due to an inflammatory exudate but in some cases they are fibrosed and then persist after death Streptococci have been grown from the pericardium from the affected heart valves and from subcutaneous nodules

**Clinical Findings** The patient is usually a child or young adult The first attack is said to occur under the age of 15 years in 90% of cases In children the onset is often insidious Many children suffer

from subacute rheumatism the chief symptom of which is so called growing pains, which come and go and are felt in the limbs or back They may also complain of attacks of abdominal pains If the patient is an adult he may have noticed an attack of acute pharyngitis 10 to 28 days previously with malaise and fleeting limb pains He is then suddenly seized with acute pain in one or more joints or in the praecordium A history of similar previous attacks may be given

*On Examination* *The patient is usually pale and not infrequently has auburn hair* *Sweating is a characteristic feature* *a sudaminal rash* may be seen and the sweat has a rather sour smell The tonsils are often *unhealthy enlarged pitted or fibrosed* *Evidence of infection* may be seen around the teeth and a dental radiogram may show apical infection Usually the larger joints are affected such as the ankles knees elbows and shoulders They may appear normal or be definitely swollen This swelling is due either to periarticular œdema or to fluid in the joints Fluid removed from a distended joint is usually sterile The skin over the joint is either normal in colour or a little red and hot and there is tenderness on palpation any attempt to move the joint causing very severe pain The changes may spread rapidly from joint to joint one improving as another is involved In children only one joint may be affected This is painful and usually swollen The temperature is usually over 100 F and rises as fresh joints are affected Afebrile periods may occur during which the pulse remains frequent The heart Rheumatic carditis *is present in almost every severe case* of rheumatic fever although clinical evidence of this may be lacking in some instances If there is no cardiac enlargement and no murmur is *heard there is no clinical evidence of cardiac involvement* *The rate* of the heart the position of the apex and the presence or absence of murmurs should be recorded daily, together with the character of the heart sounds Special attention is paid to the tone of the first sound at the apex and whether or not the second sound is reduplicated there

Rheumatic nodules are felt chiefly in children indicating that the disease is still active and they are often associated with carditis They are about ½ inch long and are found under the skin especially over the knees elbows occiput scalp backs of the hands and back of the chest The tissues should be put on the stretch to feel them They often come and go quite rapidly Erythema marginatum occurs in about 25% of cases and less often erythema nodosum

The urine is diminished high coloured and may contain a trace of *albumin* The blood *Culture is usually sterile* *There is a leuco*cytosis in the acute stages and anæmia may rapidly appear The rate of sedimentation of the red cells is increased during the active stages of the disease The antistreptolysin titre is increased and rises during the course of the disease the normal value being about 100 units

Differential Diagnosis Acute rheumatism must be distinguished from other forms of acute infective arthritis such as acute osteoarthritis and acute rheumatoid arthritis and from gout the response to salicylates in acute rheumatism is a good therapeutic test

In children acute rheumatism may be diagnosed when in reality

the illness is due to osteomyelitis in the latter the pain and tenderness are near to rather than in a joint and they are localised to one spot Scurvy and poliomyelitis, owing to the pain or immobility of the limb, may also lead to confusion, if the patient is not examined thoroughly In some cases abdominal pain and vomiting are mistaken for acute appendicitis, and an unnecessary operation performed

**Course and Complications** The course is usually rapidly modified by the administration of salicylates the temperature falling to normal in a week or 10 days There is a tendency for the pains to pass from joint to joint and for symmetrical joints to be affected Relapse may also occur with a rise of temperature and recurrence of pain in the joints previously affected The heart is the danger spot in acute rheumatism the apex beat may pass outwards a little owing to dilatation, a soft apical systolic murmur being heard due to dilatation of the mitral valve This probably indicates some myocarditis Rheumatic carditis may be definitely diagnosed if there is cardiac enlargement, a systolic murmur at or near to the apex, an accentuated pulmonary second sound and an accentuated first sound at the apex An aortic diastolic murmur also indicates carditis Pericarditis or pericardial effusion may develop Other complications include chorea, and hyperpyrexia (cerebral rheumatism) the temperature rising to over 107° F Rheumatic pneumonitis and pleurisy are rare complications The important sequelæ are cardiac lesions such as chronic endocarditis usually of the mitral valve, leading to stenosis or *affections* of the aortic valve such as regurgitation Pericardial adhesions may result from pericarditis Subsequent attacks of rheumatic fever are not uncommon No permanent joint damage results

**Prognosis** The disease is rarely immediately fatal and the type is becoming progressively less virulent in England Chorea is an unfavourable complication The ultimate prognosis depends upon the presence and severity of the cardiac lesions

**Treatment** *Prophylactic* Acute streptococcal tonsillitis should be treated with penicillin Unhealthy tonsils should be removed and the teeth well cared for damp clothes should be changed as soon as possible In order to prevent recurrence of the disease it is *advised* that a child should be given sulphadiazine 0 5 G daily for a child under 4 stones and 1 G daily for children over this weight for a period of 5 years The child must be kept under medical supervision during this time

*Curative* The patient should be put to bed between blankets or woollen sheets and kept lying down with only one pillow He must remain thus in bed until the temperature has been normal for 3 weeks after the salicylates have been discontinued providing there is no cardiac affection The sleeping pulse rate and the *sedimentation* rate of the red cells must also be normal After this additional pillows may be given and the patient gradually got up providing the pulse rate is not accelerated or the temperature raised If the heart is involved he should be kept in bed for a minimum of 3 months Procaine penicillin *300 000* units should be injected intramuscularly for 10 days *Sod salicyl gr*

Iv sod bicarb gr 30 syr aurant m 30 aq ad fl oz ½ should be given every 3 hours for 4 doses and then every 4 hours When the effect of the drug is produced as shown by relief of pains and fall in temperature the dosage is reduced to six hour intervals and then to 3 times a day The average dose required is gr 1 per lb body weight every 24 hours and the blood salicylate concentration should be kept at about mg 30 per 100 ml It is easy to raise the plasma salicylate content to the effective level but it is difficult to maintain it there as it tends to fall although the same dosage is given The dose must often be increased to prevent relapse If toxic symptoms occur such as marked buzzing in the ears nausea vomiting or hæmaturia or if the pulse falls below 60 it should be further reduced or discontinued If sodium salicylate cannot be tolerated the pure natural preparation should be tried If this disagrees salicin gr 10 to 20 may be given t d s Cortisone appears to have no superiority over salicylates in the treatment of rheumatic fever

The affected joints should be covered with ung methyl salicyl B P C wrapped in wool and protected from the pressure of the bed clothes by a cradle The bowels should be opened as necessary with aperients If there is much pain at the onset pulv ipecac et opii gr 10 should be given at night to secure sleep The diet should be fluid during the febrile stages such as barley water dextrose orangeade and milk but not meat extracts The diet is later increased with bread and butter and semi solids Subsequently the tonsils should be removed if they are septic causing enlargement of glands in the neck and repeated sore throats Removal of the tonsils will not however prevent the occurrence or re occurrence of rheumatism in children Dental sepsis should also be eradicated Owing to the prolonged rest required children are best sent after the acute stage to special recovery homes and later to open air or residential schools These should not be by the seaside or near a river Tropical or subtropical climates are most suitable

## Influenza

### (*La Grippe*)

**Definition** An acute infectious disease characterised by fever prostration a great liability to pulmonary complications and to epi demic incidence

**Etiology** The Hæmophilus influenzæ (Pfeiffer s bacillus) is not usually accepted as the cause of influenza and its role is uncertain although it is frequently found in the sputum in all types of the disease *H influenzæ is accepted as the cause of influenzal meningitis* Three viruses have been found in influenza A B and C but in many cases no virus can be detected Virus A was described in 1933 when it was found that garglings from influenza patients would produce a trans missible disease in the ferret It is more virulent than Virus B several strains exist and it is liable to cause epidemics every two years Virus B discovered in 1940 differs from A in its antigenic properties Virus C was discovered in 1949 Influenza tends to be endemic and to cause epidemics every 4 or 5 years of a comparatively mild type The majority *of wide spread epidemics have been associated with virus A infection*

During an epidemic it may be impossible to isolate a virus from as many as 75% of the cases which are clinically typical examples of influenza Pneumonic complications are probably less common in B infections Streptococci pneumococci and staphylococci are probably secondarily infecting organisms The cause of pandemic influenza, the most important variety, has not been shown to be a virus, and remains uncertain *Predisposing causes* Nothing is known on this score except that the disease tends to occur in pandemics about every 10 to 40 years and in epidemics about every 33 weeks In the 1889 pandemic infants and old people were affected whereas in the 1918 pestilence young adults in the prime of life were the victims It is estimated that 20 million deaths occurred in this pandemic Three waves were noted in the last two pandemics the second one being the most serious The disease spreads with great rapidity

**Pathology** Post mortem the changes resemble those of a hæmorrhagic septicæmia, but the spleen is not enlarged Hæmorrhages may be seen in the sheath of the rectus abdominalis and in the muscle itself The pericardium may contain a little blood stained fluid The trachea shows a typical pink colour of its lining membrane between the rings especially in the lower part The bronchi contain muco pus Exudation may be seen in the bronchioles and alveoli which prevent proper aeration of the blood Hæmorrhagic areas of lung tissue which may float or sink in water are typical with patches of collapsed lung Petechiæ may be seen under the pleuræ yellow or blood stained fluid may be found in the pleural sacs The bronchial glands are enlarged and may show hæmorrhages Petechial hæmorrhages are frequent in the mucous membrane of the stomach and the interior of the ileum cæcum or pelvic colon may be intensely congested The kidneys are usually a little enlarged and congested

**Incubation Period.** This is probably about 48 hours

**Clinical Findings** *The Mild Type* The patient can often say the hour at which he was suddenly taken ill with lassitude or severe prostration and at times shivering Pain may be felt in the head behind the eyes and on moving the eyes, under the sternum along the diaphragm attachment in the back in the calves or shins and occasionally in other parts of the body The patient may feel sick, have epistaxis or vomit

*On Examination* The conjunctivæ are often injected ('pink eye ) the face flushed and the skin dry The temperature rises rapidly to about 103° F and the pulse is moderately rapid There is usually cough on the second day and the voice may be hoarse even in mild cases careful examination of the lungs usually reveals slight abnormalities such as weak breath sounds at a base or a few basal rales or some scattered rhonchi A scarlatiniform rash may occur on the arms body and legs but the flexures are usually left clear The blood shows a leucopenia The urine usually contains no albumin The temperature falls to normal about the third or fifth day according to the type of epidemic prevailing and convalescence is rapidly reached

*Severe Cases* *The Bronchopneumonic Type* The onset is similar to

that of a mild case and it is impossible to say in any instance whether the patient will rapidly become dangerously ill The severe symptoms may be due to an acute toxæmia the temperature rapidly rising to 104° F or higher and the patient becoming cyanosed and dying in the course of a few hours In other cases bronchopneumonia or a severe purulent bronchitis may develop in a few days probably due to a secondary infection The respirations become rapid and may reach 50 to 60 a minute but the pulse is not usually proportionately frequent 110 to 120 being an average rate, and the temperature varies from 100 to 103 F or higher A peculiar heliotrope cyanosis is typical in which the face and ears are mauve but they may be leaden in colour or the face may be pale with lips and ears blue The respirations though rapid are not difficult and there is no orthopnœa The cough is often troublesome and the sputum is of varying types being either white and frothy green and purulent or tenacious and containing bright red or brown blood Examination of the lungs often shows very slight signs but generalised rhonchi may be heard or there may be small areas of dulness with weak breath sounds and crackling rales The blood is sticky the red cells may number 8 000 000 per c mm and it is difficult to bleed a patient This is commonly met with in pandemics There is usually a leucopenia of about 3 000 to 4 000 white cells per c mm with about 50 to 60% of polymorphonuclear cells The urine may contain a trace of albumin a few red and white blood cells are often found and occasionally a hyalo granular cast Fatal cases often become delirious towards the end with low muttering Patients who recover may complain of attacks of giddiness or profuse sweating during convalescence

*The Gastro intestinal Type* Other cases as I frequently observed in the 1918 pandemic assume a gastro intestinal form with vomiting and diarrhœa and bright blood may be passed in the motions

**Differential Diagnosis** A mild case of influenza is differentiated from an ordinary feverish cold by the greater prostration and muscular pains in the back limbs and external ocular muscles and also by its occurrence during an epidemic Abroad it must be distinguished from such diseases as malaria or dengue The pulmonary varieties would not usually be diagnosed unless occurring in an epidemic but the heliotrope cyanosis and tendency to hæmorrhages are suggestive

**Course and Complications** The course is very variable as described above Complications include Bronchopneumonia sterile pleural effusions empyema subcutaneous emphysema herpes facialis menin gitis sinusitis otitis media mastoiditis jaundice neuralgia and acute thyroiditis A latent tuberculous focus in the lungs may be activated and pulmonary fibrosis or bronchiectasis may ensue as a sequela Profuse generalised sweating sometimes follows an attack of influenza and persists for a week or so

**Prognosis** Mild cases recover Between 20 to 40% of severe cases develop bronchopneumonia Cyanosis is a very grave omen and cases seldom recover if the respiration rate exceeds 50 Profuse sweating is a very favourable sign in severe cases

**Treatment** *Prophylactic* During an epidemic all crowded places should be avoided The throat should be gargled night and morning with 1 in 4,000 solution of potassium permanganate Vaccination with an anti influenzal virus vaccine is of doubtful value The dose is 1 ml A preliminary desensitising dose of 0 02 ml should be given as the vaccine contains egg albumin Attendants on patients should wear a gauze mask

*Curative* The patient should be isolated in bed in an airy room at the first symptom, and remain there until the temperature has been normal for 2 or 3 days in a mild attack Returning to work too early involves a risk of a serious and perhaps fatal relapse The bowels should be opened with aperients if necessary For the muscle pains and headache aspirin gr 5 to 10 or a tablet containing aspirin gr 4 phenacetin gr 4, and codein phosphate gr 1/8 can be given t d s The diet should be fluid during the fever stage with plenty of bland drinks over 5 pints a day should be taken to eliminate toxins Linct codein (B P C), m 30 to 60 may be given t i d to relieve a dry and exhausting cough If there is severe cyanosis oxygen should be administered continuously through a nasal catheter or B L B mask At the first sign of pulmonary congestion or consolidation penicillin should be injected intramuscularly in doses of 600 000 units of a procaine preparation every 12 hours There is no specific remedy The diarrhoea is treated by giving a powder containing pulv ipecac et opii gr 5 and bismuth salicyl gr 10 three times a day

During convalescence a tonic such as Liq strychnin m 2, syr ferri phosphat co m 60 may be given t d s and no strenuous exercise should be taken which might cause the heart to dilate

## Tetanus

### *(Lockjaw)*

**Definition** An acute disease, due to a specific bacillus characterised by violent and painful muscular spasms

**Etiology** Tetanus results from infection with the Clostridium tetani an anaerobic organism which forms very resistant spores The bacilli occur in the intestines of animals such as the horse and cow Man is infected through a wound being contaminated with soil containing the spores Catgut used at an operation or wool used for dressings may contain the spores

**Pathology** The bacilli remain localised, but give rise to very potent exotoxins These are probably absorbed from the end plates of motor nerves in the muscles and pass along the nerves to the central nervous system, where they increase the excitability of the synapses Post mortem muscles such as the rectus abdominalis may be found ruptured There are very slight changes seen in the central nervous system

**Incubation Period** This is usually about 12 days *In acute cases* it may be as short as 2 days or it may be prolonged to several months especially if a prophylactic injection of antitetanic serum has been given

**Clinical Findings** The patient may give a history of a wound when gardening or there may have been a war wound or a compound fracture or he may be convalescent from an operation Tetanus is now more often seen in association with trivial than with severe wounds owing to the generalised use of antitoxin in the latter In some cases tetanus appears to arise idiopathically but the organism then presumably enters the body through an abrasion In tetanus neonatorum infection occurs through the severed umbilical cord The patient first notices muscular stiffness affecting the jaw neck or extremities In the course of a few hours the stiffness increases and painful cramps may occur in the affected muscle groups with dysphagia In children convulsions may be the first manifestation of the disease

*On Examination* In the early stages no abnormality may be found except an increase of the deep reflexes such as the knee jerks In a developed case the picture is most striking and terrible The patient has frequently recurring seizures of violent muscular contractions so that the body may be arched backwards resting on the occiput and heels (opisthotonus) or bent forwards (emprosthotonus) or twisted sideways (pleurothotonus) The jaw muscles may be firmly contracted (trismus) and contraction of the facial muscles produce the appalling risus sardonicus Trismus is rarely combined with facial paralysis Any slight external stimulus such as a noise a bright light or a touch may provoke an attack The mind remains clear and the patient may cry in agony or be unable to articulate owing to spasm of the intercostal and respiratory muscles The skin is moist or sweating but the temperature and pulse are usually normal The temperature may rise to 108° F in the terminal stages although the patient is free from spasms The blood A leucocytosis of about 12 000 per c mm may be present The cerebro spinal fluid is usually normal but under increased pressure In local tetanus the spasms are limited to a group of muscles This occurs especially some months after a bony injury In cephalic tetanus there is dysphagia and paralysis of cranial nerves together with generalised convulsions

**Differential Diagnosis** A clue to the diagnosis is usually afforded by the history Other causes of trismus (see p 388) must be excluded and spasms due to strychnine poisoning meningitis or tetany In tetanus the muscular spasm does not fully relax between the attacks

**Course and Complications** In fatal cases the spasms become more severe and frequent Complications include rupture of a muscle and bronchopneumonia

**Prognosis** Early and efficient treatment render the outlook more hopeful but even then the mortality is usually over 50% Death may occur in 3 or 4 days from asphyxia or heart failure The prognosis is usually worse if tetanus rapidly follows the infliction of a wound Cole calls the period of onset the time between the onset of trismus and the first generalised reflex spasms A period of onset of less than 2 days indicates a very grave prognosis The prognosis is better with wounds of the lower extremities

Treatment *Prophylactic* Tetanus toxoid 1 ml given subcutaneously

and repeated in six weeks, is used for prophylactic inoculation of members of the fighting forces Subsequently, in the 1939–45 war an annual dose of 1 ml was given to all soldiers Any wounded man who had not been so immunised was given 3 000 units of antitoxin at weekly intervals a single dose of 3 000 units being given to an immunised wounded man Antitetanic serum should be given in any case of a wound which may have been contaminated by soil The prophylactic dosage for an adult is 500 units subcutaneously repeated three times at weekly intervals Larger doses are required if there are septic wounds *i e* 3 000 to 5 000 units every other day for 3 doses and then weekly injections for 3 weeks or until the wound is healed The wound should also be disinfected with 1 in 20 carbolic acid solution

*Curative* The patient should be nursed on a water bed in a dark, quiet room A cradle should be used to keep the bedclothes away from his body A single large intravenous injection of 200 000 international units of antitetanic serum should be given after testing to see whether the patient is sensitive to horse serum The effect of this will last for 10 days No advantage is gained by intrathecal or cisternal injection of serum Some authors recommend in addition the subcutaneous injection of 1 ml of tetanus toxoid at the first onset of symptoms This is repeated at intervals of 5 or 6 days After an interval of at least an hour the wound should be opened, syringed with hydrogen peroxide and a light gauze dressing applied which will not exclude the air This dressing is repeated every 4 hours If there are no reflex spasms phenobarbitone gr 1 should be given by mouth every 4 hours Mental distress and reflex spasm should be relieved by the intravenous injection of Pentothal in a 2 5% solution The amount required to produce the required sedation and the frequency of the injections can only be determined by the effect achieved The needle should be kept in the vein A muscle relaxant drug is also required such as *d* tubocurarine chloride 30 mg /ml in an oil and wax base It is injected intra muscularly in doses of 0 5 to 1 ml or if a more rapid effect is required it is given intravenously in saline 3 mg /ml The dose is usually 3 mg /40 lb body weight Tracheotomy may be necessary at any moment A special nurse is required day and night to feed the patient A dietary containing at least 2 000 calories daily is necessary, in the form of dextrose lemonade eggs and milk If there is profuse sweating 1 to 2 pints of normal saline should be given by mouth or by rectum If the patient cannot swallow he should be fed through a nasal tube passed into the stomach which should be left in position between the feeds

## Actinomycosis

**Definition** Specific granulomatous lesions caused by a mycotic organism

**Etiology** The cause is the Actinomyces bovis (streptothrix actinomyces or ray fungus) Wolff and Israel showed this to be a strict anaerobe The aerobic streptothrix of Bostroem which is found

in barley and grasses, is probably non pathogenic The so-called actinomycotic lesions in cattle such as woody' tongue and lumpy jaw, are usually due to an aerobic organism the actinobacillus lignieresi but some of these lesions are due to the actinomyces bovis Grains and grasses are thought to cause abrasions through which the infecting organisms enter Man is probably infected by the organism entering damaged mucous or cutaneous surfaces The most frequent portals of entry are the teeth tonsils appendix and the skin Males are chiefly affected between the ages of 20 and 40

**Pathology** The granulomata break down to form abscesses containing little pus which often discharge through multiple sinuses Yellowish sulphur granules are present in the pus consisting of a central mycelium and peripheral clubs The lesions occur most frequently in the head and neck They may be found in the lungs pleura or chest wall, in the appendix cæcum rectum or liver in the kidneys or female genital tract or in the skin A blood borne infection may result in cerebral lesions

**Clinical Findings** The onset is usually insidious and the findings differ widely according to the site of the lesion

*Cervico-facial Type* This is a surgical condition Swellings occur in the face jaw bone or neck which gradually soften with multiple sinus formation

*Thoracic Type* This is described on p 178 The clinical picture in the early stages may resemble that of bronchitis or of pleurisy later an empyema, pulmonary tuberculosis syphilis or a new growth of the lung may be suggested

*Abdominal Type* The lesion usually spreads from the appendix to the cæcum Secondary liver abscess may develop or an abdominal swelling may form resulting in sinuses through the abdominal wall It is often only detected at an operation for appendicitis'

*Genito urinary Type* The clinical picture is that of salpingitis or ovaritis, or rarely of a suppurative renal lesion

*Cutaneous Type* The skin and subcutaneous tissues alone may be involved Possibly infection occurs here from straw

*Nervous Type* Infection may spread from the naso pharynx along the olfactory nerves to the brain causing an isolated lesion near the pituitary or the fornix Direct spread may occur from the jaw to the base of the brain or a cerebral abscess or meningitis may follow a pulmonary lesion

*Vertebral Type* The vertebræ may be affected secondarily to a focus elsewhere Collapse of the body of a vertebra may ensue

**Differential Diagnosis** The condition may closely simulate sarcoma tuberculosis or a pyogenic infection The diagnosis is established by bacteriological examination of the pus a special request should be made to this effect when the material is sent to the laboratory

**Course and Complications** The course is a chronic one Complications are rare and are due to the infection being blood borne to other sites such as the liver and brain

**Prognosis** This is favourable in the cutaneous and cervico facial

types, especially if adequate treatment is given early It is more unfavourable in the deep seated cases

**Treatment** The pus should be evacuated surgically if possible Potassium iodide should be given by mouth in gradually increasing doses up to gr 90 three times a day The patient is usually tolerant of large doses Crystalline penicillin should be injected intramuscularly in doses of 4 to 8 million units daily, in divided doses, for about 6 weeks This may be combined with sulphadiazine Deep X ray treatment is of value in some cases

## Sarcoidosis

### (*Besnier Boeck Schaumann Disease*)

**Definition** Sarcoidosis is a chronic usually benign, lympho granulomatosis of widespread distribution in the body

**Etiology** The cause is unknown The view that sarcoidosis is due to tuberculosis is not generally accepted

**Pathology** Typical granulomata form consisting of central giant cells surrounded by epithelial cells with an outer layer of lymphocytes *Caseation and calcification do not occur* The skin mucous membranes and most of the organs of the body may be affected

**Clinical Findings** Various types of sarcoidosis are met with according to the location of the lesions Thus the first symptom may be mistiness of vision due to irido cyclitis There may also be enlargement of the parotids sometimes accompanied by facial palsy This constitutes the uveoparotid fever of Heerfordt Hæmorrhages may occur in the vitreous secondary to periphlebitis of retinal veins (Eale's disease)

In other cases the patient complains of vague symptoms of ill health or at a routine mass miniature X ray examination of the chest bilateral enlargement of the hilar glands—the so called potato glands—is seen Other pulmonary abnormalities revealed by X ray examination include fine mottling coarse infiltration fibrosis collapse and cystic changes

Erythema nodosum is met with in about one third of the cases and there may be nodular skin eruptions especially on the butterfly area of the face the nose ears and fingers which constitute lupus pernio Bone lesions are found in about 15% of cases X ray examination showing punched out areas of rarefaction in the phalanges the *osteitis tuberculosa multiplex cystica* of Jungling There may also be general atrophy of the bones of the hands and feet Sarcoid nodules may be found in the tonsils Hypercalcæmia with renal failure is met with in some cases *possibly due to sarcoid lesions in bones* The liver and spleen may be enlarged and lymphatic glands palpable in front of, and behind the ears in the axilla groins neck and elsewhere

The total plasma proteins are often increased with a reversal of the albumin globulin ratio due to increase of globulin The Mantoux test is usually negative even to a dilution of 1/100 The Kveim test consists in the intradermal injection of an emulsion of a sarcoid gland A nodule forms at the site of the injection in about 4 to 6 weeks if the

test is positive On biopsy this shows a typical sarcoid lesion If no superficial gland is available for biopsy the diagnosis may be established by liver puncture the sarcoid being demonstrated in the fragment of tissue removed even when the liver is not enlarged

**Prognosis** This is usually good the lesions spontaneously disappearing or healing by fibrosis When the eyes are affected the outlook is more serious Pulmonary fibrosis may lead to congestive heart failure In some cases tuberculosis supervenes The mortality rate is about 5%

**Treatment.** In cases in which the patient s *health* is affected cortisone should be given by mouth in doses of mg 200 daily This is gradually reduced as improvement is obtained Eye lesions should be treated by an ophthalmologist

## Glandular Fever

### (*Infectious Mononucleosis*)

**Definition** An acute infectious disease characterised by glandular enlargement fever and excess of mononuclear cells in the blood

**Etiology** The cause is unknown It may be due to an ultra microscopic filterable virus *Predisposing causes* 1 Age Children and young adults of both sexes are especially affected 2 Season It tends to occur in the winter and spring and there may be small epidemics

**Pathology** The glands show lymphoid hyperplasia with loss of their normal structure changes closely resembling those seen in lymphatic leukæmia There is lymphocytic infiltration of the spleen and rupture sometimes occurs The liver is frequently enlarged and granulomatous lesions may be found in the bone marrow

**Incubation Period** This is usually 7 to 8 days

**Clinical Findings** The patient is usually a child or young adult who may not feel well for a few days before being definitely taken ill He then complains of headache fever pain in the neck and perhaps soreness of the throat and vomiting

*On Examination* Three types are described —The glandular the anginose and the febrile *The glandular type* The tongue is furred and the fauces are red Enlarged glands may be seen and felt in the neck and under and behind the sterno mastoid muscles These are usually unilateral but both sides are affected later Torticollis may be present for a few days Enlarged glands may also be found in the submaxillary region in front of the ear in the axillæ and in the groins They are firm and a little tender and the patient usually holds the neck rather stiffly The temperature is raised to about 103 F or over and the pulse is proportionately rapid Enlargement of the bronchial glands may result in a paroxysmal cough and there may be abdominal pain if the mesenteric glands are affected The spleen may be felt in about one third of the cases Jaundice rarely occurs at times during the course of the disease less often it is the presenting symptom before there is any apparent glandular enlargement In the few cases recorded it has usually been obstructive in type but occasionally the van den

Bergh reaction is indirect and bile is present in the fæces In the latter cases the jaundice cannot be due to pressure of enlarged glands in the portal fissure *The anginose type* A membrane is seen on or near the tonsils resembling that of diphtheria The cervical glands are not as large as in the glandular type There may be œdema of the neck with considerable local tenderness The temperature often remains raised for 2 to 3 weeks *The febrile type* This affects chiefly adults A pink maculo papular rash appears, chiefly on the trunk between the fourth and seventh days Glandular enlargement is not usually noted until the end of the third week Relapses may occur for many months Various neurological manifestations have been noted including headache, encephalitis, meningitis ataxia coma, convulsions hemiplegia, ocular and facial palsy peripheral neuritis and paralysis of the serratus magnus The cerebro spinal fluid may show a lympho cytosis The blood White cells There is a leucocytosis of about 20,000 per c mm 80 to 90% are mononuclear cells either immature lymphocytes, large or small lymphocytes or large mononuclears The red cells are not usually affected In some cases there is no leucocytosis and the mononuclears are not more than about 40% The blood changes may also be of very short duration The Wassermann reaction is positive during the second and third weeks in about half the cases Paul and Bunnell state that the blood serum agglutinates sheep's red cells in a dilution of 1 in 64 by the fourth day of the disease and that this test is diagnostic Other authorities do not consider a titre of less than 1 in 256 is diagnostic unless the absorption test is also positive A posi tive Paul Bunnell reaction may also be given in serum sickness, but the heterophile antibody present in these cases can be distinguished from that of glandular fever by absorption tests The urine Albumin and blood may be present

**Differential Diagnosis** Glandular fever is differentiated from mumps as the parotid gland is not involved Other conditions which have to be excluded are Vincent s angina, diphtheria leukæmia Hodgkin s disease, and enlarged glands due to tuberculosis syphilis German measles, typhoid fever or sepsis The white cell count and glan dular enlargement may suggest acute leukæmia but the absence of changes in the red cells is of great significance The cough and blood count may also suggest whooping cough Appendicitis may be simulated if the abdominal glands are affected The course of the disease usually renders the diagnosis clear Early jaundice and fever may suggest infective hepatitis or cholecystitis In agranulocytic angina there is leucopenia, with over 90% of lymphocytes and an almost complete absence of granular cells

**Course and Complications** The temperature usually falls to normal in 1 to 2 weeks but may remain slightly raised in the evening for 6 months, the glands may remain palpable for several weeks A relapse may occur about 4 weeks after the onset, with fever and enlargement of glands Suppuration is rare. Hæmorrhagic nephritis may occur

**Prognosis** The disease is rarely fatal although death may occur from bronchopneumonia in the anginose type

**Treatment.** The patient should be in bed for 2 weeks there is no specific treatment The throat should be swabbed with $H_2O_2$ (10 vols) and cataplasma kaolini applied to the neck During the acute febrile stage the patient is kept on a fluid diet and the bowels should be opened daily

## Glanders

### (*Farcy*)

**Definition.** A specific granuloma caused by a definite bacillus

**Etiology** Glanders is caused by the Pfeifferella mallei The disease is transmitted to man by direct contact with an infected animal such as a horse donkey or mule the organisms entering an abrasion in the skin or nasal mucous membrane It may also be conveyed from man to man

**Pathology** The bacilli form an endotoxin mallein A granuloma results from the local inoculation and septicæmia or pyæmia may ensue The nasal lesions constitute glanders the subcutaneous nodules are called farcy buds and the enlarged lymphatics farcy pipes Abscesses may be found post mortem in the liver and spleen

**Incubation Period** This varies between 3 days and 3 weeks

**Clinical Findings** *Acute glanders* The patient is usually a worker amongst horses He complains of malaise headache and at times of nausea and vomiting

*On Examination* The temperature is raised to 101 F or more and runs an irregular course The initial lesion may be seen on the hand, arm or face as a papule surrounded by a red area The lymphatics may stand out as red streaks a generalised papular or pustular eruption may then appear and abscesses form subcutaneously or intramuscularly In other cases lesions also occur in the nose and are accompanied by a sticky exudate ulceration of the nose and enlargement of the cervical glands Pneumonic and rheumatic forms of acute glanders are also described Glanders bacilli may be isolated from the nasal discharge the abscesses or the sputum *Chronic glanders* This is characterised by the appearance of subcutaneous nodules generally on the forearms which ulcerate and have a purulent discharge Constitutional dis turbance is slight

**Differential Diagnosis** The diagnosis is suggested by the patient s occupation The lesions must be differentiated from other granulo mata such as actinomycosis or syphilis and the generalised pustular eruption from small pox The bacteriological findings establish the diagnosis Melioidosis a disease of rats cats and dogs in Rangoon and Indo China occasionally affects man and is very similar to glanders It is caused by the P pseudomallei

**Course and Complications** Acute glanders is usually fatal in under 2 weeks Chronic cases may persist for months and then gradually abate or they may suddenly become acute

**Prognosis** The acute variety is practically always fatal in the chronic form the outlook is much more favourable

**Treatment** *Prophylactic* Animals suffering from glanders should be destroyed

*Curative* Some favourable results have been obtained by treatment with sulphadiazine sulphadimidine and streptomycin The patient must be kept in bed and abscesses opened as they form

## Anthrax

(*The Malignant Pustule Wool sorter's Disease*)

**Definition** An acute specific infective disease which may assume a cutaneous *pulmonary or intestinal form*

**Etiology** Anthrax is caused by the Bacillus anthracis which forms *spores and thus exists for long periods in a viable stage* outside the body Animals, such as sheep, cattle goats horses and reindeer after eating *infected grass, suffer from* splenic fever *Man is infected on the skin* (the malignant pustule) by handling infected hides of animals or by using infected shaving brushes usually made from horse hair The pulmonary variety of anthrax results from inhaling spores or bacilli in woollen occupations (wool sorter's disease) and possibly the intestinal form is due to eating infected meat

**Pathology** The organisms are often present in the blood stream, causing a septicæmia in wool sorter's disease the bronchial glands are enlarged subpleural hæmorrhages may be present and areas of œdema or collapse are seen in the lungs with bronchitis In the intestinal variety hæmorrhagic areas may be seen in the mucous membrane of the bowel with thrombosis of mesenteric veins Meningitis may be present as a complication in any septicæmic form of the disease

### The Malignant Pustule

**Incubation Period** *This is usually less than 24 hours*

**Clinical Findings** Infection occurs through an abrasion in the skin The patient may be a *worker in hides* or be infected *through* a shaving brush Itching is first noticed on the skin of the face arm or neck and a small red papule then appears at the site of *the itching* which quickly becomes inflamed and angry looking The patient may feel quite well up to within a few hours of his death or be *very ill with* malaise, shivering and headache He suffers little or no pain in the lesion

*On Examination* The papule is seen encircled by whitish *vesicles*, and in a day or so a black slough forms in the centre The tissues around become brawny and the neighbouring glands *enlarge* The temperature may be normal *or* raised to about 103° F In severe cases the blood culture is positive *before* death, which occurs *in about* a week from the onset

**Differential Diagnosis** *The malignant pustule must be differentiated* from a septic spot or boil, a chancre or a subcutaneous lesion in glanders The appearances are very suggestive in anthrax, *the history is usually* confirmatory and anthrax bacilli are found in the serum from the vesicles

**Prognosis** *The pustule may disappear spontaneously* If the

case is treated efficiently within a day or so of its onset the hope of recovery is very good

Treatment. *Prophylactic* All shaving brushes should be free from anthrax spores

*Curative* The pustule should not be excised as this increases the risk of septicæmia It should be covered with gauze to absorb exuding serum and the affected part of the body kept absolutely still by splints or sandbags The use of Sclavo s serum has now been largely replaced by aureomycin and terramycin

### Anthrax Œdema

A diffuse œdema of a limb the face or body may occur without any local pustule being seen

### Wool sorter's Disease

*(Pulmonary Anthrax)*

**Clinical Findings** The patient is usually suddenly taken ill with malaise and shivering followed by pain in the chest, cough expectoration and much weakness

*On Examination* The appearances are those of a very severe illness the temperature is usually over 103° F the pulse and respirations are rapid The lungs show signs of generalised bronchitis The sputum is frothy and blood stained and may contain anthrax bacilli The blood culture is usually positive before death The disease is usually fatal within a few days

Treatment This is as described above

### Intestinal Anthrax

This is a rare variety The clinical picture resembles that of acute food poisoning with abdominal pain vomiting and diarrhœa The spleen may be palpable and anthrax bacilli are found in the fæces It is usually fatal

Treatment This is as described above

### Hydrophobia

*(Rabies La Rage Lyssa)*

**Definition** A disease caused by the bite of certain rabid animals

**Etiology** The virus is ultramicroscopic and filtrable It is present in the saliva of mad dogs cats or wolves Vampire bats were probably the carriers of the virus in an outbreak in Trinidad Man is infected by a bite or by the rabid animal licking a raw surface on his body The disease is practically non existent in England now having been eradicated by the orders for the muzzling of dogs and later by the quarantine laws which are still enforced

**Pathology** Negri bodies are found in the brain of rabid animals They occur in nerve cells especially in the hippocampus major and cerebellar cortex They are the inclusion bodies of rabies

**Incubation Period** The average is 6 weeks, but it may vary from 12 days to 8 months or longer

**Clinical Findings** The patient may be a child or adult who gives a history of being bitten by a rabid animal, usually a dog The bite heals but at the end of the incubation period symptoms are noted The disease falls into three stages *The invasion* Irritation or pain is felt at the site of the bite and the patient becomes irritable or depressed and does not feel well The voice may be a little hoarse The temperature is found to be slightly raised *The stage of excitement* The patient is now acutely ill and in a pitiable condition he is very restless and is seized with painful muscular spasms affecting the muscles of deglutition and respiration The spasms are provoked by swallowing, or even by the sight or thought of food or water or by any sudden stimulus

*On Examination* The temperature is raised to over 101° F, and the pulse is frequent Saliva may run from the mouth and mucus from the nose the patient is cyanosed during the spasms and may be maniacal (*la rage furieuse*) This stage lasts for 2 to 8 days and then passes into *The stage of paralysis* The patient is now exhausted and muscular paralyses develop The temperature falls there is unconsciousness, and death from heart failure occurs in a few hours

*Varieties* *La rage mue* This is rare in man There is no excitable stage the patient is quiet and is rapidly paralysed and dies

**Differential Diagnosis** The diagnosis is established by the history of the bite of a rabid animal, the typical clinical findings and examination of the brain of the animal which shows Negri bodies If the suspected dog is alive 10 days after the bite the diagnosis of rabies is excluded Rabies must be differentiated from —Pseudohydrophobia This is a hysterical condition occurring after a dog bite There are no true spasms, but the patient may bark or bite like an animal Tetanus or strychnine poisoning The history here is suggestive and the muscles of deglutition and respiration are not involved in the manner typical of hydrophobia Acute bulbar paralysis Here there is no history of a bite and the maniacal symptoms do not occur Acute poliomyelitis It may be impossible to differentiate this during life as in the epidemic in Trinidad in 1931

**Course and Complications** The disease is rapidly fatal lasting 4 to 5 days and there are no complications

**Prognosis** Multiple bites are more serious than solitary ones, and bites on the head and face are followed by a more rapid onset than those occurring distally from the central nervous system Hydrophobia is more likely to follow bites of wolves than those of dogs The disease is invariably fatal if not checked during the incubation period

**Treatment** *Prophylactic* The importance of muzzling dogs in endemic zones and of quarantining imported dogs has been mentioned A dog bite should be allowed to bleed freely and then immediately washed with 1 in 1 000 perchloride of mercury solution and cauterised with fuming nitric acid The patient should then be treated with a vaccine consisting of a suspension of infected rabbit s or sheep s brain

inactivated by ultra violet light 2 ml are injected daily for 14 days If given early rabies can be prevented in the vast majority of cases

*Curative* Only palliative measures can be employed such as injections of morphine or inhalations of chloroform

## Psittacosis

### (*Ornithosis*)

**Definition.** An acute infectious disease conveyed by parrots and allied birds and birds of other orders characterised by fever prostration and often by pulmonary symptoms

**Etiology** It is now generally accepted that the disease is due to a filterable virus Epidemics occur chiefly in houses and flats In July 1929 there was a fairly extensive epidemic in the Argentine and cases occurred in England in the autumn and winter of 1929–1930 probably due to parrots imported from South America Budgerigars (love birds) may also transmit the disease In 1934 red cockatoos (galahs) and budgerigars imported from Australia to England were found to be infected In Germany in 1934 over 150 cases of psittacosis occurred in six months The source of infection was found to be apparently healthy budgerigars who are carriers of the virus the virus probably having been imported into the country by parrots before 1931 The virus is thought to be present in the bird s fæces and beak discharges Pigeons canaries finches ducks and chickens may also convey the infection Infection of man is probably through the respiratory tract Direct infection from man to man is uncommon

**Pathology** The spleen is a little enlarged red and soft The lungs Petechial hæmorrhages may be seen under the pleura and areas of consolidation occur which are dull red and dry with no evidence of suppuration Microscopically characteristic elementary bodies are found in the hilar glands alveolar exudate Kupffer cells spleen and lymph glands

**Incubation Period** This is about 10 days

**Clinical Findings** The patient is usually an adult who gives a history of contact with a parrot or budgerigar which was ill or has subsequently died or with a budgerigar apparently in good health which is a carrier of the virus The onset is somewhat sudden with malaise shivering headache and at times severe epistaxis nausea vomiting or diarrhœa

*On Examination* At the onset the temperature is raised to about 102 to 104 F but the pulse is slow under 100 The patient may be drowsy or complain of severe occipital headache The temperature remains raised for 1 to 3 weeks and gradually falls by lysis A cough often appears after a few days and examination of the lungs shows signs of bronchitis This may be followed insidiously by the development of areas of consolidation or collapse in the lungs which disappear as the temperature falls X ray examination shows patchy shadows in the lungs In some instances the patient is overwhelmed with toxæmia he has a low muttering delirium and a Parkinsonian like expression

is very obvious In other cases there is much abdominal distention with offensive loose motions Small red spots may appear on the chest or back The spleen is rarely palpable The urine often contains albumin The blood The white cells usually number about 7 000 per c mm but *in some cases a leucocytosis has been recorded The blood* may contain the virus during the first seven days of the disease as shown by mouse inoculation A complement fixation test using as an antigen 5% virulent mouse spleen in phosphate or saline, is used as a diagnostic test and a mouse test can also be *employed, the animal being* infected by the patient's sputum or pleural fluid

**Differential Diagnosis** The disease is diagnosed chiefly on circumstantial evidence and must be differentiated from influenza, primary atypical pneumonia, enterica infections miliary tuberculosis pneumonia or Bact coli urinary infection The onset is more sudden than in enteric fever There are neither eye nor limb pains as in influenza The urine does not contain the colon bacillus The history of contact with a sick parrot or budgerigar the presence of the typical clinical picture described above and the demonstration of the virus in the bird or the patient and the complement fixation test enable the diagnosis to be made

**Course and Complications** The average course is 3 weeks

**Prognosis** The disease is serious the mortality varying between 10 to 30% It is worse for old people

**Treatment** *Prophylactic* The sick bird should be destroyed by coal gas or chloroform and the carcase burned taking care that the operator wears rubber gloves which are also burned *The danger* from healthy budgerigar carriers still exists in this country

*Curative* The patient should be kept in bed until the temperature has been normal for 4 days Terramycin or aureomycin should be given in doses of 1 G every 8 hours for the first 2 days and then 1 G every 12 hours for 7 days

## Coccidiomycosis

**Etiology** Coccidiomycosis is caused by the fungus *Coccidiodes immitis* It occurs especially in California Arizona and Texas Man is usually infected by inhalation and at times through the skin

**Pathology** The fungus produces nodules which may form abscesses in the bones subcutaneous tissues lungs liver and spleen, and less often in the kidneys brain, meninges peritoneum and pericardium

**Clinical Findings** Mild subclinical cases may occur but the patient is usually acutely ill with fever and prostration The symptoms closely resemble those of pulmonary tuberculosis

*On Examination* Signs of bronchopneumonia may be found in the lungs which may lead to thin walled cavities Skin lesions resembling those of erythema multiforme may appear These constitute the ' Valley fever ' or San Joaquin fever ' Dissemination may occur with involvement of bones and internal organs The causative fungus may be found in the sputum and the complement fixation test becomes positive

**Prognosis** This varies with the location of the disease It is good in the primary pulmonary type but very grave when the disease becomes disseminated

**Treatment.** No specific treatment is required for primary pulmonary cases Potassium iodide and the use of a coccidiodal vaccine are recommended for the disseminated variety

## Toxoplasmosis

This disease is caused by a protozoon the toxoplasma first described in the North African gondi (a small rodent) in 1909 It has subsequently been found in many places in Europe and in North and South America and in other animals birds and reptiles In 1939 a case was described of acute fatal encephalitis in a child due to toxoplasma The disease may be congenital or acquired and it is met with clinically in an acute subacute and chronic stage In the congenital form the symptoms of the acute illness are due to encephalomyelitis In the subacute variety there may be progressive encephalomyelitis hydrocephalus choroido retinitis cerebral calcification of granulomata and convulsions Very similar changes are met with in the chronic form The acquired disease usually manifests itself by symptoms of encephalitis by febrile exanthematous states associated with pneumonitis or necrotic glomerular lesions in the kidneys or the infection may be latent The liver or spleen may be enlarged and swelling of the lymph glands may suggest a diagnosis of glandular fever In all active cases the prognosis is very grave

The diagnosis may be confirmed during life by a complement fixation test or by a dye test of the serum which inhibits the toxo plasma protozoon from staining with methylene blue Sulphadiazine in full doses appears to have some value but the antibiotics are ineffective

## Histoplasmosis

This disease is caused by a fungus the histoplasma capsulatum Man is infected by the inhalation or ingestion of the spores They are found *in man intracellularly in the cells of the reticulo endothelial* system, in the lungs the sputum the blood the bone marrow etc It resembles coccidiomycosis in appearing as a mild or primary form or in a severe disseminated form It is endemic in the valleys of the Mississippi and Ohio rivers in the St Lawrence area in Mexico and Panama It has also been met with in South America Australia and Northern Europe

In the acute pulmonary form the signs and symptoms resemble those of diffuse pneumonitis In only a few cases does the disseminated variety occur There is then often fever diarrhœa enlargement of lymph glands and of the spleen and liver Involvement of the bone marrow causes anæmia and leucopenia Lesions may also occur in the skin the tongue the larynx etc The histoplasma skin test performed by injecting intradermally 0 1 ml of a 1/1 000 dilution of histoplasmin solution is positive and a positive complement fixation test can also be

obtained No specific cure has been discovered for the disseminated variety the primary pulmonary variety requires only symptomatic treatment

## Spirochætal Jaundice

*(Spirochætosis Icterohæmorrhagica Leptospiral Jaundice Weil s Disease)*

**Definition** A disease characterised typically by jaundice and hæmorrhages due to a spirillum

**Etiology** Weil s disease is caused by the Leptospira *icterohæmorrhagiæ* a coarse spirillum, whose dimensions are $12\mu \times 0.25\mu$ It is present in sewer rats (Rattus norvegicus) less commonly in the black rat (Rattus rattus) and is excreted in their urine water thus becoming infected The L canicola is carried by dogs, and man may be infected Transmission to man is probably through the skin possibly by contaminated food, or by swallowing infected water while bathing in canals *Predisposing causes* Locality Spirochætal jaundice occurs in Holland Japan Egypt the Malay States especially in mines, and was met with during the 1914–18 war in France and Gallipoli Outbreaks have also occurred in Great Britain amongst sewer workers tripe and fish cleaners coal miners and farm workers

**Pathology** The body is usually jaundiced Hæmorrhages may be seen in the muscles skin, central nervous system mucous membrane of the stomach and duodenum, and in the lungs and kidneys The kidneys often show tubular degeneration The liver may appear normal or fatty The spleen is usually soft and in the type described by Weil it is enlarged

**Incubation Period** This is usually 8 to 12 days

**Clinical Findings** The onset is usually sudden and there may be a rigor The patient complains of headache and severe pains in the limbs and back There may be marked giddiness and vomiting or diarrhœa Milder cases also occur

*On Examination* The conjunctivæ are injected the temperature is high, about 103° F and the pulse comparatively slow, 80 to 90 Jaundice appears in about half the cases beginning on the third or fourth day when the skin may itch Petechial hæmorrhages may occur and bleeding from the gums or nose and in some cases there is labial herpes which may be hæmorrhagic Blood may be brought up from the stomach or lungs or passed in the motions The temperature remains irregularly raised and falls by lysis reaching normal about the seventh to fourteenth day It may subsequently rise again about the sixteenth day for a few days The tongue is dry The spleen is not usually palpable but the liver may be felt The glands in the axillæ and groins may be enlarged The motions are usually constipated and pale The blood The leptospira is present for the first week of the illness and may be demonstrated either by blood culture or by intraperitoneal injection of the blood or urine into the guinea pig A blood count shows a hæmolytic anæmia with a leucocytosis of 20,000 to 30 000

per c mm The platelet count is low The serum after the sixth day will agglutinate formalised cultures of the leptospira in a dilution up to 1/30 000 An agglutination of 1/400 or over is considered to be positive and a rising titre indicates an active infection The use of muscle biopsy has been recommended as an early diagnostic test before the agglutination reaction becomes positive As shown by Pick there is loss of striation and vacuolation is seen in striated muscle fibres The blood urea is often raised The urine is scanty and contains albumin bile and blood and the leptospira may be present after the tenth day Jaundice rarely occurs in L canicola infections but meningitis or meningismus is not uncommon and often severe

**Differential Diagnosis.** The disease must be differentiated from jaundice due to enterica infections and from yellow fever Other conditions such as infective hepatitis and relapsing fever must be excluded Cases in which there is no jaundice are liable to be mistaken for influenza The diagnosis depends upon finding the leptospira in the blood or urine on a positive agglutination test and the leucocytosis

**Course and Complications** Some cases pursue a severe course with intense jaundice and meningitis Meningitis may occur apart from jaundice and the onset may be delayed for several months after the beginning of the disease The diagnosis is established by finding the leptospira in the cerebro spinal fluid and urine by guinea pig inoculation Iritis may occur as a complication Death may occur from hepatic toxæmia and uræmia

**Prognosis** The disease is a serious one but the large majority of cases recover

**Treatment** *Prophylactic* This is concerned with the destruction of rats and the wearing of shoes by miners in infected areas

*Curative* The patient should be put to bed and the bowels opened with a saline aperient The diet should be liquid avoiding fats as for infective hepatitis Dextrose orangeade should be given and alkalis in doses sufficient to render the urine alkaline Penicillin must be given in very large doses up to 5 million units every 24 hours to have any beneficial effect Aureomycin may be tried in doses of 1 G every 4 hours Antispirochætal serum is also available 20 ml may be given intravenously three times a day for 3 days in a severe case It should be given within the first 3 to 5 days later it has little effect The fæces and urine must be disinfected as for enteric fever

## Tularæmia

(*Rabbit Fever Deer fly Fever*)

**Definition** A disease characterised by fever and glandular enlargement due to a specific bacillus

**Etiology** Tularæmia is caused by the Pasteurella tularensis The disease occurs in squirrels rabbits water rats and sheep It is conveyed to man by the deer fly (*Chrysops discalis*) Man may also be infected in preparing dead rabbits for eating and in laboratory work Locality

Tularæmia is met with in Tulare County, California in other parts of America Japan, Russia Norway France Belgium Germany Turkey etc

**Pathology** Small areas of focal necrosis are found in various organs in the body

**Incubation Period** This is usually between 1 and 10 days

**Clinical Findings** *The Glandular Type* A local papule appears at the site of the fly bite This ulcerates and the neighbouring lymphatic glands enlarge There is an irregular temperature, which may last for 2 or 3 weeks The blood A positive agglutination test may be obtained towards the end of the second week

*The Typhoid Type* When laboratory workers are infected the disease assumes the character of a septicæmia There is irregular fever lasting for 2 or 3 weeks and relapses may occur for several months The blood culture is usually positive

Other types include the ophthalmic pleuro pulmonary oral and abdominal

**Differential Diagnosis** The disease has to be distinguished from other septicæmias and causes of glandular enlargement with fever, such as the enterica group and plague Injection into a guinea pig of the fluid obtained by gland puncture is a method of isolating the bacillus Other diagnostic measures include an intracutaneous an agglutination and a complement fixation test

**Course and Complications** The course is as described above Bronchitis bronchopneumonia pneumonia or pleural effusion may occur as complications

**Prognosis** The average mortality rate is about 5% but it is higher in the pulmonary variety

**Treatment** The most effective treatment is streptomycin injected intramuscularly in doses of 0 5 G morning and evening for 12 days In the pleuro pulmonary variety the dosage should be 2 G twice daily

## Cat Scratch Disease

*(Cat bite Fever)*

This resembles rat bite fever The causative organism has not been isolated Two or three weeks after the bite or scratch of a cat the regional lymph glands enlarge there is a relapsing fever and a macular rash appears on the forehead or trunk The disease is quickly cured by the intravenous injection of 0 3 G neoarsphenamine or by the oral administration of aureomycin

## CHAPTER X

# THE LOCOMOTOR SYSTEM

## THE MUSCLES AND FASCIÆ

### Fibrositis

### (*Muscular Rheumatism Myalgia*)

**Definition** Inflammation of the connective tissue of muscles, fasciæ ligaments nerve sheaths tendons and periosteum

**Etiology** This is uncertain There is no proof that it is due to infection in the intestine or in other sites such as the teeth tonsils sinuses or gall bladder Exciting causes are strain cold and damp climates

**Pathology** Small nodules are formed in some cases in the muscles lipomatous masses between the superficial and deep fascia which are vascular and become œdematous These congested and swollen fatty masses tend to bulge through fibrous layers and to become strangulated They are easily palpable They constitute trigger points for referred sciatic pain and inflammatory changes occur in the connective tissues

**Clinical Findings** The patient is usually an adult There may be a history of cold exposure or commonly of muscular strain The lumbar muscles are often affected (lumbago) or those of the neck (stiff neck) or chest (pleurodynia) An attack of lumbago may come on quite suddenly on stooping or on making a violent movement with the leg or arms such as braking or cranking a car In the subacute cases there is a less severe pain felt in the back which is intensified on stooping lifting sneezing or coughing On deep palpation the nodules which are very tender can usually be felt A stiff neck (torticollis) usually follows sitting in a draught one or other sternomastoid or trapezius muscle being affected The intercostal muscles may be involved causing pleurodynia which is usually unilateral

**Course and Complications** In lumbago the inflammatory process may extend to involve the sheath of the sciatic nerve with consequent sciatica Generally the patient recovers from an acute attack in 3 to 4 weeks but he is liable to recurrences

**Treatment** During the acute stage rest and warmth are essential In acute lumbago an injection of procaine 1% in normal saline into the tender nodules or just beneath the surface of the fascia covering the affected muscles often gives rapid relief of pain and freedom of movement Two to 10 ml are injected at each site up to a maximum of 30 ml After the injection the patient is instructed to put the affected muscles through a full range of movement and this should cause no pain The patient should only remain in bed if the pain is very severe and cataplasma kaolini may be applied to the back every 12 hours

during the acute stages Aspirin gr 10 t i d or tab codein co 1 t i d or tab pethidine hydrochlor mg 50 by mouth may be given for the relief of pain In very severe cases it may be necessary to inject morphin sulph gr ¼ intramuscularly at the site of the pain Subsequently any focus of sepsis should be eradicated and care taken that the bowels are completely evacuated daily, as there is often some degree of constipation present All strains to the back should be avoided and occupations involving stooping are most unsuitable

## Epidemic Myalgia

### (*Epidemic Pleurodynia Bornholm Disease*)

**Etiology** This is a comparatively rare disease of epidemic nature occurring chiefly in the summer months and affecting principally children and young adults It was first described in Norway in 1872 and later in the Island of Bornholm in the Baltic in 1930 In 1944 there was an outbreak in Alabama and cases have occurred in England Three small outbreaks were recorded in England in 1949 and 1950 There is evidence that some epidemics are associated with infection by Coxsackie virus 1 or 2 or very closely allied viruses Coxsackie is a village in New York State where the infection was first described in patients thought to be suffering from non paralytic poliomyelitis summer grippe or epidemic myalgia

**Clinical Findings** The incubation period is thought to be 2 to 4 days The disease is characterised by a sudden onset of pain around the diaphragmatic attachments often unilateral The pain is intensified by coughing and sneezing Pains may also occur in the abdomen back or neck and frontal headache is a characteristic feature The temperature is raised to about 104° F for 24 to 48 hours Dry pleurisy may be present and some cases have been described under the title of acute benign dry pleurisy in the Middle East during the 1939–45 war The disease must be differentiated from an acute abdominal lesion especially when there is vomiting and abdominal distention It may also resemble influenza but diaphragmatic pleurisy is not a characteristic feature of the latter There is a tendency for a relapse to occur 2 or 3 days after the temperature has fallen to normal In patients affected with the Coxsackie virus complement fixing antibodies are found in the serum by the third day and they show a rising titre The virus has also been recovered from the throat and fæces of patients suffering from the disease

**Treatment** This consists in rest in bed a bandage round the lower ribs to diminish diaphragmatic movement and aspirin for relief of pain

## Dermatomyositis

**Definition** A disease characterised by inflammation and degeneration of muscles subcutaneous œdema and various forms of dermatitis It is classified as a collagen disease (see p 637)

**Etiology** This is unknown

**Pathology** The muscles are œdematous pale red or yellow Areas

of round celled infiltration are seen in the muscles with atrophy of the muscle fibres and thinning of the epidermis Hæmorrhages may occur (*Polymyositis hæmorrhagica*)

**Clinical Findings** The onset is usually insidious with pain or cramps in the muscles anorexia malaise and pyrexia In some cases there may be dysphagia diplopia or difficulty in breathing

*On Examination* Œdema of the face and eyelids may give the appearance of alabaster Generalised œdema may occur Various erythematous skin rashes may be seen Occasionally there is eosinophilia The spleen is enlarged and various muscles are tender weak wasted and often stiff The diagnosis may be established by muscle biopsy

**Prognosis** Death occurs in about 60% of cases and may result from involvement of the muscles of respiration

**Treatment.** The patient must be kept in bed massage and electrical treatment may be used and the pain relieved by analgesics such as aspirin In some cases the basal metabolic rate is low and good results are obtained from the administration of thyroideum starting with gr ½ daily and gradually increasing the dose Temporary remissions are obtained in some cases by cortisone or ACTH

## Primary Myositis Fibrosa

*Etiology* This is unknown

**Pathology** The muscle fibres atrophy and are replaced by connective tissue

**Clinical Findings** The patient notices swelling and pain in the muscles of the legs

*On Examination* There is little tenderness but the affected muscles are very hard and subcutaneous œdema may be found over them

**Prognosis** The disease often spreads until nearly all the voluntary muscles are affected

**Treatment** Massage and electrical treatment may be tried and improvement often results

## Progressive Myositis Ossificans

**Etiology** The cause is unknown

**Pathology** Embryonic connective tissue is first formed this is converted into fibrous tissue and later ossification occurs Deposits of bone are found in the muscles tendons ligaments and fasciæ and there are exostoses Some cases follow trauma and are preceded by a hæma toma

**Clinical Findings** The patient is usually a young adult men are attacked more frequently than women The early symptoms suggest muscular rheumatism and at the onset there may be slight fever with some redness or swelling of the skin over the affected muscles usually the back and the neck Bone formation gradually occurs in the muscles and spreads to the ligaments causing fixation of joints The muscles of mastication may be involved with fixation of the jaw Severe scoliosis may ensue Exostoses are frequently noted on the humerus

tibia fibula and ribs The big toes and thumbs are deformed and small the interphalangeal joint being ankylosed and the metacarpal or metatarsal bone stunted

**Prognosis** This is very unfavorable and death usually occurs from some intercurrent infection after the patient has become bedridden

**Treatment** There is no curative treatment good nursing care is required in advanced cases

Other affections of voluntary muscles which are mentioned under their particular sections include Trichiniasis (see p 756) Degenerations (such as Zencker s degeneration see enteric fever p 579) Hæmorrhage as in scurvy or in influenza (see p 602) into the rectus abdominalis Rupture, as in tetanus Tuberculosis A cold abscess in muscle may be due to direct spread from a bone abscess rarely in miliary tuberculosis foci are found in the voluntary muscles In syphilis a gumma may form

## The Muscular Dystrophies

*(The myopathies and muscular diseases of doubtful nature)*

In this group of cases the primary changes are regarded as muscular rather than nervous The characteristic features of the myopathies are — A familial history The age of onset Different muscle groups are affected in different types Pseudo hypertrophy occurs in some cases There are no fibrillary contractions such as occur in cord lesions There is no reaction of degeneration and no sensory changes occur, as in neuritis

### Pseudo hypertrophic Muscular Dystrophy

**Etiology** The cause is unknown The disease tends to run in families being transmitted by the mother Boys are usually affected

**Pathology** There is atrophy of the muscle fibres with increase in the fat and connective tissue of the affected voluntary muscles causing apparent enlargement There are no changes in the central nervous system

**Clinical Findings** The patient is usually a boy aged 8 to 12 years There is a history that he began to walk late and that he has been unsteady on his legs and finds stairs difficult

*On Examination* The general condition is good and there is enlargement of the muscles of the calves (gastrocnemius and soleus) the front of the thighs (quadriceps extensor and sartorius) the buttocks (glutei) the infraspinatus triceps and at times the deltoid The enlarged muscles are found to be weak The patient stands on a wide base lordosis is marked the gait is waddling and in order to rise from a supine posture the patient climbs up himself ' first rolling over on to his hands and knees and then working his hands up his legs There is usually wasting of the latissimus dorsi and lower part of the pectoralis major muscles so that if the child is lifted up under the arms he tends to slip through the hands The face with the exception of the masseter muscles and the forearms are not affected There are no sensory changes The deep reflexes in connection with the affected

muscle groups are gradually diminished and there are no fibrillary tremors Electrical reaction The reaction of degeneration is not present but the response to faradisation and galvanism gradually diminishes

**Course and Complications** The disease is progressive and death usually occurs before adult age from wasting or secondary infections

**Treatment** There is no curative treatment and it is doubtful whether vitamins or glycine are of any value Massage and exercises are usually beneficial

### Juvenile Muscular Dystrophy

*(Erb s Dystrophy)*

**Etiology** The cause is unknown

**Pathology** There is wasting of the fibres of the affected muscles

**Clinical Findings** The patient is usually between the ages of 15 and 35 male or female and several members of a family may be affected *Weakness is first noticed in the arms or legs and the muscles* are later found to be wasted

*On Examination* The muscles wasted are usually those of the upper arm (biceps triceps and supinator longus) and the thigh (glutei extensors and hamstrings) giving a bottle-shaped appearance to the limbs Some of the trunk muscles may also waste such as the latissimus dorsi lower part of the pectoralis major trapezius rhomboids serratus magnus erector spinæ etc The face is not usually affected Lordosis is present the electrical reactions and deep reflexes are diminished The reaction of degeneration is not present and there are no sensory changes and no fibrillary tremors In some instances a distal type (of Gowers and Spiller) may be seen in which the forearms hands and wrists legs feet and ankles and muscles of the face are wasted

**Course and Complications** The disease is usually progressive but the patient may live until middle age before being carried off by some intercurrent infection

### Facio Scapulo Humeral Dystrophy

*(Landouzy Déjerine s Dystrophy)*

This is a variation of the juvenile type which occurs usually in infancy has the same hereditary factor and affects the sexes equally

**Clinical Findings** Weakness and wasting are first noticed in certain face muscles These are the orbicularis oris and orbicularis palpebrarum the eyelids cannot be closed and the lips are everted the lower lip projects (tapir mouth) On smiling the lips are straight The shoulder girdle muscles including the trapezius latissimus dorsi serratus magnus, pectorals triceps and biceps are subsequently affected The scapulæ become winged

**Course and Complications** The course is progressive but the patient may live until adult life

## Amyotonia Congenita or Myatonia Congenita

### (*Oppenheim s Disease*)

**Etiology** This is unknown The disease resembles a myopathy in many respects

**Pathology** The anterior horn cells of the spinal cord are usually diminished in number especially in the lumbar region and the corresponding anterior nerve roots are thin and deficient in myelin The muscle fibres on the whole are small, but a few abnormally large ones are found The connective tissue and fat of the muscle are increased

**Clinical Findings** The disease may have a familial incidence. The onset is at or before birth and the condition is characterised by extreme flaccidity of the voluntary muscles an infant if sitting tends to fall forwards and resembles a frog (Batten s frog child) The muscles are very weak and the child may be unable to hold up its head The legs are more severely affected than the arms The hands and feet are long and narrow The face is not affected The deep reflexes are absent and the electrical response to faradisation is diminished but the reaction of degeneration is not present

**Differential Diagnosis** Amyotonia congenita is differentiated from poliomyelitis by the general distribution of the paresis and absence of complete paralysis in amyotonia congenita and from the myopathies by the absence of marked wasting and the tendency to improvement Its resemblance to the Werdnig Hoffmann disease has been noted on p 440

**Course and Complications** The child may soon die from intercurrent infection but there is a tendency to improvement with an increase of muscle tone and recovery of the deep reflexes so that he may survive and reach adult life

## Myotonia Congenita

### (*Thomsen s Disease*)

**Etiology** The cause is unknown

**Pathology** The voluntary muscle fibres may be increased in width There is no increase in the muscular connective tissue and there are no nervous changes The muscular contractions resemble those produced experimentally with veratrin there being delay in contraction and relaxation Experimental evidence on goats suffering from a similar disease suggests that the lesion is a primary muscular one and not due to changes in the myoneural junctions

**Clinical Findings** The disease is very rare occurring more commonly in males tending to run in families and first showing itself in childhood The patient notices a stiffness in the voluntary muscles chiefly in the hands arms feet and legs and to a lesser degree in the trunk The muscles of mastication may also be affected

*On Examination* It is found that the voluntary contraction and relaxation of the muscles is prolonged but a movement becomes more

speedy after repetition This can be well tested by asking the patient to grip the hand several times There is no spasticity found on passive movement The skeletal muscles are usually enlarged The sensation is normal The following distinctions exist between this condition and the myopathies The deep reflexes are not affected there is no atrophy of muscles the electrical reaction is peculiar, the faradic and galvanic responses are first small and prolonged but on repeated stimulation they become normal (myotonic reaction) there is a modified reaction of degeneration as A C C approximates to K C C (normally K C C > A C C and in the reaction of degeneration A C C > K C C)

**Course and Complications** The disease has no effect on the duration of life

**Treatment.** Quinine sulphate or hydrochloride gr 5 to 10 once to thrice daily should be given by mouth the dose being increased until the myotonia is relieved An adequate maintenance dose must be worked out

## Myotonia Atrophica

*(Dystrophia Myotonica)*

**Etiology** The cause is unknown

**Pathology** There is atrophy of the majority of fibres in the affected muscles with some giant fibres

**Clinical Findings** The patient is usually a male, aged between 20 and 35 There is often a family history of the disease and also of cataract The onset is insidious with pains in the arms or legs weakness loss of weight and a difficulty in relaxing the grip

*On Examination* There is wasting of the muscles of the face the neck (sterno mastoids) the forearms and the legs and the testicles may be atrophied The deep reflexes in connection with the affected muscle groups are diminished There are no sensory changes no fibrillary contractions and no reaction of degeneration Electrical stimulation causes a prolonged relaxation after contraction

**Course and Complications** The course if untreated is slowly progressive Death usually occurs from intercurrent disease

**Treatment** Quinine should be given as in myotonia congenita

## Myasthenia Gravis

**Etiology** The cause is unknown The disease appears to be due to a chemical abnormality (deficiency of acetyl choline) which results in a defect of transmission of the impulse from the nerve to the muscle Acetyl choline is the chemical substance which allows transmission of impulses across the myo neural junctions in striated muscles but acetyl choline itself has no beneficial effect in the disease It is thought that eserine and neostigmine (Prostigmin) inhibit the destruction of acetyl choline by an esterase It has been suggested that a hypothetical inhibitor substance is produced in the thymus which circulates in the blood stream and interferes with the transference of cholinergic impulses

from nerve to muscle and that this substance is neutralised by Prostigmin

**Pathology** Lymphorrhages" (small round cells) are found between the muscle fibres There are no primary changes in the nervous system The thymus is frequently enlarged containing many lymphoid germinal centres and a thymoma may be present

**Clinical Findings** The disease affects men and women equally and usually begins in adults before middle age, but it may be met with in young children The patient may complain of inability to keep his eyes open or of diplopia which increases during the day and disappears temporarily after a night s rest In other cases varying fatigue is noticed in different muscles causing difficulty in speech or swallowing alteration in voice or weakness of the neck or limbs Thus if the arm is affected, the performance of any movement involving its use will cause rapid fatigue, power being regained temporarily after a short rest Aching in the limbs may also be noticed

*On Examination* In a typical case certain muscles supplied by the cranial nerves are affected especially those of the eyelids and the external oculo-motor muscles, with resulting ptosis strabismus and inability to close the eyes tightly Monocular myasthenia gravis has been described The lips may also be involved so that the patient cannot whistle or smile normally If the palate is affected it rapidly fatigues when the patient is asked to say Ah The voice may be nasal and nasal regurgitation of fluids occurs on swallowing The tongue and jaw muscles may also be involved with dysarthria and dropping of the jaw In other cases there is weakness of the neck so that the head is supported on the hands or fatigu ability of the arms may be demonstrated for when the patient holds the arms extended they gradually droop to the sides The muscles are not usually wasted the deep reflexes are variable the jaw jerk may be abolished but the knee jerk is not lost The electrical tests show the myasthenic reaction the response to faradisation is rapidly exhausted but is restored by rest Galvanic stimulation does not fatigue the muscles and they will contract voluntarily when they do not respond to faradisation Sensation is usually unaffected The urine The creatine content is high and creatinine low

**Differential Diagnosis** The characteristic expression of the patient with drooping eyelids and elevated head and the fatigu ability of the muscles are typical Other causes of ptosis must be excluded such as hysteria tabes dorsalis and cerebral lesions Organic bulbar paralysis and diphtheritic neuritis must be differentiated in some cases

**Course and Complications** The course is progressive but may be interrupted by remissions especially during pregnancy Involvement of the respiratory muscles may cause death

**Treatment** This consists in rest for the muscles and tonics Electrical treatment should not be given Beneficial results were obtained in 1929 with ephedrine hydrochloride in doses of gr ½ to 1 t i d

and in 1932 with ephedrine gr ⅜ together with glycine (glycocoll) oz ¼ twice daily In 1934 a further advance was made by using neostigmine (Prostigmin) a synthetic physostigmine If neostigmine methyl sulphate 1 to 2 5 mg and atropin sulph gr 1/100 are injected sub cutaneously the patient recovers power in the affected muscles in about 10 minutes and the effects are maintained for about 3 to 6 hours To produce comparable results by oral administration 15 mg of neostigmine bromide are equivalent to an injection of 1 mg A severe case may require as many as twenty 15 mg tablets of neostigmine bromide spaced out through the 24 hours In each case the maintenance dose must be worked out To prevent abdominal pains tinc belladon m 20 should be taken as required Thymectomy has been successful in many instances Keynes obtained a complete or almost complete remission of symptoms in 65% of his cases The operative mortality rate is less than 5% in skilled hands It does not appear that the result of the operation depends upon the condition of the thymus whether it is normal hypertrophied or contains a thymoma although the presence of a thymoma renders the operation more dangerous Good results have also been obtained in some cases by radio therapy when the thymoma has been demonstrated by lateral tomography

## Familial Periodic Paralysis

*(Intermittent Myoplegia)*

**Etiology** The cause is unknown The disease tends to run in families

**Pathogenesis** The paralysis appears to be connected with the amount and form of potassium salts in the muscle fibres It has been shown that an attack can be produced in a susceptible individual by the administration of dextrose 200 G by mouth This results in the fall of the blood potassium paralysis usually appearing when the figure is below 10 to 12 mg per 100 ml (normal 17 to 22 mg per 100 ml )

**Clinical Findings** The disease usually first shows itself in childhood or about the age of puberty There are recurring attacks of paralysis which may affect the arms legs trunk and neck Usually the face eyes sphincters and diaphragm are not involved In some cases only the lower limbs are affected and the weakness may be greater in one half of the body than the other Premonitory symptoms are sometimes noted such as lassitude tingling in a limb hunger thirst palpitations or sweating A heavy meal may predispose to an attack The onset usually occurs during sleep the patient on waking finds that he cannot move his limbs The attack usually passes off in a few hours or in a day or so

*On Examination* The affected muscles are flaccid and completely or partially paralysed The cutaneous and deep reflexes are diminished or abolished The electrical reactions are diminished or absent Sensa tion is usually normal The blood potassium falls during an attack

**Differential Diagnosis** Owing to the rarity of the condition and the transitory nature of the attacks the patient runs a risk of being diag

nosed as suffering from hysteria or malingering The condition must also be differentiated from *potassium losing nephritis*, which may be met with in patients suffering from chronic pyelonephritis This usually first shows itself in a later age group than does familial periodic paralysis In potassium losing nephritis the attacks are of longer duration and usually more severe hypopotassæmia and muscular weakness tend to persist between the attacks In the absence of albuminuria tests of tubular function such as the urine concentration test and the ability to acidify the urine, will usually show evidence of renal failure

**Prognosis** The attacks tend to become less severe and frequent after middle age

**Treatment** The patient should avoid a high carbohydrate diet which lowers the serum potassium. The attack can usually be relieved by administering at the onset pot chlorid gr 90 to 180 for an adult and gr 15 to 30 for an infant, dissolved in 2 fl oz of water and taken in small quantity of milk

## THE BONES

### Osteitis Deformans

### (*Paget's Disease*)

**Definition.** A chronic disease of bone characterised by enlargement of the skull kyphosis and bowing of the extremities

**Etiology** The cause is unknown The disease is almost always associated with atheroma

**Pathology** The pelvis spine, skull tibia or clavicle is generally first affected The changes are those of subperiosteal new bone formation and deeper areas of rarefaction There is much bony thickening and irregularity The skull is thickened with narrowing of the foramina and sutures (see Fig 59) The bone fat is increased and its calcium diminished The rate of blood flow through the bones may be very considerably increased

**Clinical Findings** The patient is usually a male aged 40 to 60 and the disease tends to run in families The onset is insidious pains may be first noticed in the legs (shins) or enlargement of the head or bending of the bones may be the first symptom *Examination of a Developed Case* The patient presents a typical appearance the calvarium is enlarged and the face appears triangular with the base upward (see Fig 60) The legs and arms are bowed forwards and outwards Kyphosis is marked, usually in the upper dorsal region and the spine is very rigid There is also thickening and enlargement of the pelvis, shoulder girdle tibiæ and femurs and to a lesser degree of the bones in the arms The patient's height may be reduced by 4 to 6 inches but the hands and feet are usually unaffected In rare cases the changes are limited to a single bone such as the clavicle, the ischium, pubis, ilium or one vertebra The disease is then said to be monostotic and is discovered accidentally on X ray examination In these cases the alkaline serum phosphatase may not be increased In the majority of



FIG 59 SKULL IN PAGET'S DISEASE

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cases extensive arteriosclerosis is noted The skin over the affected bones may be red and hot Narrowing of the foramina in the skull may compress the cranial nerves and give rise to optic atrophy oculo motor paresis deafness and perhaps facial palsy (see Fig 60) X ray examination of the bones shows the enlargement and deformity and areas of rarefied spongy bone and of dense amorphous bone are seen giving a woolly appearance to the skull The blood The serum calcium and phosphorus are normal The alkaline phosphatase is usually increased *The sedimentation rate of the red cells is likely to be normal* The urine calcium content is usually increased in the early stages later it may be normal or diminished It has been shown that if the affected bone is immobilised bone destruction continues without bone formation There is then an excess of calcium in the blood and increased calcium excretion in the urine If the kidneys fail to excrete the calcium, death may occur from hypercalcæmia A similar effect may be produced by keeping the patient in bed

**Differential Diagnosis** There is usually no difficulty in distinguishing Paget s disease from acromegaly rickets and localised over growth of bone due to other causes such as trauma or syphilis The X ray findings are characteristic Secondary carcinoma of bone may give rise to difficulty but the primary growth can usually be found and the X ray appearances differ The sedimentation rate of the red cells is increased in carcinoma

**Course and Complications** The course is progressive the patient may live for 20 to 30 years after the onset gradually becoming more crippled and dying from an intercurrent disease Congestive heart failure may result from increased blood flow through the affected bones the circulatory disturbance being analogous to an arteriovenous shunt Fracture of the long bones compression paraplegia or osteosarcoma may occur

**Treatment** There is no specific treatment Osteotomy is not usually advisable but division of the periosteum may relieve pain Analgesic drugs such as aspirin may be required for the pain In the early stages the diet should be rich in calcium and phosphorus and vitamin D 10 000 units and vitamin C 1 000 units (mg 50) taken daily X ray treatment may relieve the pain for 2 to 3 months A low calcium diet with plenty of fluid by mouth or intravenously should be given if the patient is confined to bed and the blood calcium is excessively high

## Generalised Osteitis Fibrosa

*(of von Recklinghausen Diffuse Fibrocystic Disease of Bone)*

**Definition** A disease characterised by softening of bones formation of fibrous tissue and benign giant celled tumours and cysts

**Etiology** The disease is due to hyperparathyroidism

**Pathology** The bones are depleted of calcium The giant celled tumours consist of osteoclastic cells Some fibrosis occurs in the subperiosteum bone and bone marrow and there is local formation of new bone A parathyroid tumour is present usually an adenoma of



FIG 60 PAGET'S DISEASE SHOWING LEFT FACIAL PALSY

one or a few bones with a definite tendency to spontaneous arrest The bone lesions are frequently unilateral It occurs chiefly in adolescents and there may be spontaneous fracture Pigmentation may occur on one side of the body and premature sexual development may have been noted earlier in life The blood calcium and plasma alkaline phosphatase are normal and the disease is not associated with a parathyroid tumour or with hyperparathyroidism

### Leontiasis Ossea

A condition of hyperostosis of all the bones of the skull including the face bones The cause is not known but in some cases it may be associated with chronic sinusitis It occurs usually about the age of puberty and is more common in women than in men The patient complains of pressure effects such as headache neuralgia deafness blindness and insomnia Death may not occur for 30 to 40 years and may be due to intercurrent diseases or to a convulsion

### Osteoporosis

Osteoporosis is an extremely common disease The bone matrix is composed of nitrogenous substances In osteoporosis there is a disorder of protein metabolism resulting in a deficiency in the rate of formation of bone matrix This may be due to a reduction in activity of osteoblasts

Several varieties are described 1 *Disuse* Absence of movement removes the stresses and strains which stimulate osteoblastic activity 2 *Post menopausal* Due to lack of œstrogens 3 *Senile* Due to lack of androgens or œstrogens or both 4 *Nutritional deficiencies and glandular disturbances* Insufficient protein intake may cause a deficiency of nitrogenous supplies to the bones Osteoporosis may also be due to lack of vitamin C hyperthyroidism diabetes mellitus Cushing s disease or to the administration of ACTH or of cortisone 5 *Congenital* Fragilitas ossium is a disease of unknown etiology 6 *Idiopathic* Osteoporosis may follow a comparatively trivial accident or a normal pregnancy 7 *Localised* Osteoporosis may occur in such conditions as Sudeck s atrophy of the bones of the hands

From the clinical standpoint the senile and post menopausal varieties are most important They cause spinal osteoporosis The spine the pelvis and the neck of the femur are specially affected The centre of the vertebra becomes bi convex the cod fish vertebra The body of a vertebra may collapse and fracture of the neck of the femur may occur The serum calcium phosphorus and alkaline phosphatase readings are normal The patient loses height sometimes as much as 4 or 6 inches and a transverse crease forms across the upper abdomen

**Treatment** After the age of 60 years regular yearly records should be kept of an individual s height In this way spinal osteoporosis will be recognised in an early stage The diet should be rich in protein and when the disease is present male and female sex hormones should be administered by mouth methyl testosterone mg 10 and dienœstrol mg 1 daily These should be given for 4 weeks omitted for 2 weeks

the principal cells Rarely there is a generalised hyperplasia of all the parathyroids Calcification may occur in the kidneys

**Clinical Findings** The patient is usually over the age of 20 of either sex, more often a woman She complains of pains in the back pelvis or limbs Spontaneous fracture of a bone may occur There is tenderness on pressure over the affected bones The blood The serum calcium may be as high as 23 mg per 100 ml (normal 9 to 11 mg ) and the phosphorus as low as 1 mg per 100 ml (normal 2 5 to 5 mg ) The alkaline phosphatase is usually increased (normal 3 to 13 King Armstrong units) The urine contains an excess of calcium The calcium in the fæces is usually about normal In some cases a parathyroid tumour may be felt or it may only be discovered by an exploratory operation X ray examination shows the characteristic bony changes diminished density and pale areas due to cysts

**Differential Diagnosis** The case may be mistaken for one of rheumatism arthritis or osteomalacia With secondary malignant deposits in bone the blood calcium may rise but the phosphorus then usually rises too In some cases renal symptoms predominate either due to the formation of calcium phosphate calculi or to renal insufficiency Frequency of micturition may suggest diabetes insipidus The diagnosis is established by the X ray and blood changes and by finding a parathyroid tumour the removal of which arrests or cures the disease Fragility of bones also occurs as a familial disease associated with blue sclerotics and progressive deafness The blueness of the sclerotics is due to the dark pigment of the choroid showing through the unduly transparent sclerotics The deafness results from otosclerosis The disease usually shows itself in the second or third decade

**Course and Complications** The course is usually progressive, unless adequately treated death occurring from an intercurrent disease such as a pulmonary embolus renal failure or exhaustion

**Prognosis** This is good if the parathyroid tumour is removed early In long standing cases no increase in the density of the bones may occur for several years if at all although the progress of the disease is arrested

**Treatment** An exploratory operation should be made in the neck and a parathyroid tumour searched for and removed The tumour may lie in an abnormal site behind the œsophagus, in front of the second and third thoracic vertebræ or in the thorax Subsequently the fall in the blood calcium is controlled by giving a diet rich in calcium and by the administration by mouth of tab calcium lactate gr 60 t i d and vitamin D 50 000 units daily given as 1 tab calciferol fort if there are symptoms of tetany If the symptoms are acute 10 to 30 ml of 10% calcium gluconate should be injected intravenously daily until the blood calcium rises to normal (see p 695)

### Focal Osteitis Fibrosa

*(Osteitis Fibrosa Disseminata)*

This is a condition resembling the general disease but affecting only

cod liver oil Haliverol or calciferol sunlight and a diet rich in calcium and phosphorus such as milk, eggs fish and green vegetables In Milkman s syndrome, and in vitamin D resistant cases of osteomalacia calciferol should be injected intramuscularly in doses of 600 000 units 3 times a week Calcium lactate should be given by mouth in doses of gr 60 t i d

### Multiple Myelomatosis

*(Kahler s Disease)*

This disease is characterised by the formation of reddish tumours in the bone marrow anæmia and Bence Jones protein in the urine Spontaneous fractures may occur The tumours are composed of hæmopoietic cells A variety localised to one bone is known as a plasmocytoma The patient complains of aches or pains in the pelvis or chest, and there may be paraplegia The blood shows a leuco erythroblastic anæmia *i e* a hypochromic anæmia with normoblasts The total number of leucocytes is not increased but a few myelocytes and myeloblasts are present The serum globulin is usually increased and the sedimentation rate of the red cells is rapid The differential diagnosis includes Paget s disease Ewing s sarcoma secondary malignant deposits in bone and syphilis of the bones The X ray appearances of the bones the blood count the presence of Bence Jones protein in the urine and the result of sternal puncture which shows plasma cells in the bone marrow serve to establish the diagnosis The prognosis is hopeless but X ray treatment to the bones may relieve the pains Urethane has been recommended as relieving pain and improving the general condition It may be prescribed as Urethane gr 15 (1 G ) syrup aurant m 20 aq chlorof ad fl oz 1 The usual dose is 4 to 6 G daily for 3 weeks and then 2 G for a week or so It is discontinued if there is leucopenia Cortisone mg 300 a day in divided doses for 7 to 10 days may relieve the pain

### Diffuse Osteosclerosis

*(Albers Schonberg Disease Marble bone Disease)*

The bones become unduly rigid owing to thickening with loss of the marrow cavity The ossification probably starts before birth and fractures are not uncommon X ray examination shows a homogeneous density of bone due to deposition of calcium in the medulla The marble bone is soft and can be cut with a knife The upper and lower edges of the vertebral bodies may be affected giving a characteristic appearance (see Fig 61 p 640) The liver and spleen are enlarged The blood may show a leuco erythroblastic anæmia

### Osteogenesis Imperfecta

*(Fragilitas Ossium)*

A congenital disease characterised by extreme brittleness of the bones multiple fractures occurring before or after birth It is sometimes

and then repeated again for 4 weeks and so on A pint of milk a day should be drunk which contains mg 680 of calcium Overdosage with calcium or vitamin D is not required and may be harmful

## Osteomalacia

**Definition** A deficiency disease characterised by softening of bones with a liability to fracture

**Etiology** Osteomalacia may be due to several causes 1 Deficiency of calcium and vitamin D in the diet and absence of sunlight The blood calcium is low and there is a negative calcium balance It appears to be an adult variety of rickets This is the cause in the majority of cases 2 Deficient absorption of calcium and vitamin D in idiopathic steatorrhoea 3 Increased excretion of calcium in the urine as in the Fanconi syndrome (see p 678) 4 Following removal of a parathyroid tumour in generalised osteitis fibrosa 5 Milkman s syndrome This is by some considered to be a variety of osteomalacia Osteomalacia is endemic in Northern India Japan and Northern China Males are occasionally affected

**Pathology** The bones are brittle owing to lack of calcium The bones especially affected are those of the pelvis spine thorax and the long bones

**Clinical Findings** The patient is usually a woman between the ages of 20 and 30 who complains of weakness and aching in the back chest or legs Deformity is usually produced especially in the pelvis which interferes with childbirth there may also be kyphoscoliosis chest deformities coxa vara and bending of the long bones A spontaneous fracture of a long bone may occur The X rays show that the bones are rarefied In severe cases the bone markings disappear and the bones may not cast a more dense shadow than the surrounding tissues The plasma phosphorus and at times the serum calcium figures are below normal The clinical findings in idiopathic steatorrhoea have been described on p 65

*Milkman s syndrome* may or may not be a variety of osteomalacia X ray examination shows incomplete pseudofractures involving the flat and tubular bones They often occur at the site of pressure of an artery as where the circumflex scapular artery winds round the axillary border of the scapula Paired bones are often symmetrically involved There is no bending or bowing of the bones as is liable to occur in late rickets or osteomalacia The softening begins in the cortex and entirely encircles a tubular bone extending into the marrow cavity There is no attempt at repair The blood chemistry is normal The patient has often a waddling gait and complains of pain in the affected bones

**Differential Diagnosis** Osteomalacia is characterised by the bony changes in the pelvis and is to be differentiated from osteogenesis imperfecta generalised osteitis fibrosa and *osteitis deformans*

**Course and Complications** Tetany may occur as a complication

**Prognosis** A complete cure can usually be effected with adequate treatment

**Treatment** The patient should be treated as for rickets with

swelling of the wrists and ankles and an intermittent hydrarthrosis may occur in other joints such as the knee The changes are chiefly in the soft tissues and by X rays subperiosteal thickening may be seen

## THE COLLAGEN DISEASES

Collagen forms the basis of bone cartilage and interfibrillary connective tissue Fibroblasts are largely composed of collagen In the conditions grouped under this heading the collagen in various sites undergoes fibrinoid degeneration The degenerated tissue may be rapidly replaced by fibrous tissue as in the formation of Aschoff bodies in the heart muscle in rheumatic fever or it may be invaded by leucocytes and eosinophils as in polyarteritis nodosa It has been suggested that this change may be due to infection to allergy or according to Selye to stress According to this theory animals react to stress or to injury by what is called the general adaptation syndrome In this process it is postulated that hormones are discharged from the pituitary to act on the suprarenals The suprarenals then secrete mineralo corticoids regulating electrolytic metabolism and gluco corticoids influencing carbohydrate metabolism The value of grouping together the collagen diseases is strengthened by the fact that cortisone and ACTH cause a remission of symptoms in patients suffering from certain of them The diseases usually included in this group are rheumatoid arthritis rheumatoid lung disease rheumatic fever disseminated lupus erythematosus polyarteritis nodosa serum sickness scleroderma and dermatomyositis Other diseases which respond to cortisone are asthma Sjogren s disease sarcoidosis gout Simmonds disease and Addison s disease

## THE JOINTS

### Rheumatoid Arthritis

*(Atrophic Arthritis)*

**Definition** Inflammatory and degenerative joint changes of doubtful etiology

**Etiology** In some cases the disease appears to be related to an infective focus at the roots of teeth in the nose throat cranial sinuses bronchi intestine or genito urinary tract *Predisposing causes* 1 Age Chiefly between 20 and 45 2 Sex Females predominate 3 Exposure to cold and damp The disease is practically unknown in the tropics 4 Fatigue overwork and mental strain 5 Dietetic and endocrine deficiencies Selye produced arthritis in adrenalectomised rats by injections of desoxycorticosterone Hench drew attention to the remissions which occur during pregnancy or in jaundice There is no definite evidence that the disease is due either to a pituitary or adrenal disturbance There is however some disturbance of steroid metabolism Progesterone is excreted mainly as pregnanediol When progesterone is given to a patient suffering from rheumatoid arthritis an abnormally high amount of pregnanediol is excreted

**Pathology** Rheumatoid arthritis is considered to be one of the

associated with otosclerosis and blue sclerotics There are often protuberances on the skull and the limbs are short The cause is unknown The serum calcium, plasma phosphate and calcium excretion are usually normal

## Osteopsathyrosis

### (*Lobstein's Disease*)

This closely resembles osteogenesis imperfecta, but the fragility of the bones is not noticed until childhood or later It often runs in families

## Achondroplasia

A congenital disease characterised by arrested development of the bones of the extremities with consequent dwarfism The head is large the bridge of the nose depressed, and the hands are small with fingers of equal length Lordosis is present with contraction of the pelvis The skin over the body is thick, the voluntary muscles are powerful and the mentality is normal Males and females are equally affected

## Oxycephaly

### (*Craniofacial Stenosis*)

This rare congenital disease is characterised by deformity of the skull the vertex being pointed and forehead sloping together with the occurrence of exophthalmos and optic atrophy It is considered to be due to premature union of the sagittal and coronal sutures Digital impressions formed by bony trabeculæ are seen post mortem on the inner aspect of the vault of the skull which during life give a beaten silver appearance on X ray examination The patient complains of headaches, failing vision and possibly loss of smell Decompression, or King s mosaic operation in which the vault of the skull is separated into small squares may be required to relieve the pressure

## Hypertrophic Osteoarthropathy

A condition of ' clubbing of the fingers (Hippocratic fingers) or toes often associated with enlargement of the wrists or ankles

**Etiology** The cause is not known impeded venous return œdema and toxæmia may be factors It is usually found associated with chronic diseases of the lungs and pleura such as fibroid tuberculosis, bronchiectasis and empyema with mediastinal tumours and congenital disease of the heart In some instances no cause can be found It may develop in a few weeks in such conditions as an empyema Unilateral clubbing may occur

**Pathology** Clinically there are two varieties the drum stick with bulbous extremities to the fingers associated with bronchiectasis and the parrot bill or puffin beak variety in which the nails are curved from base to tip This is often met with in tuberculosis In the early stage the first sign is congestion of the finger tips at the roots of the nails In more advanced cases there is enlargement of the hands and feet and

thin smooth and glossy In the infective type the joint lesions may be complicated by tenosynovitis and bursitis especially around the shoulders elbows, wrists knees and ankles Fixation of the elbows and marked deformity of the hands may prevent the patient from *feeding or attending to himself or lesser degrees of crippling may exist* The blood pressure is low The epitrochlear and other glands are frequently enlarged the spleen may be palpable and there is irregular fever during the acute stages The sedimentation rate of the red cells *is increased in accordance with the activity of the disease* There is usually no leucocytosis but secondary anæmia is common Agglutination of the hæmolytic streptococcus occurs in many cases X ray examination usually *shows decalcification of the carpal metacarpal and* phalangeal bones cartilage may be eroded on the interphalangeal joints or generalised decalcification may be present Mental depression and irritability are often marked

**Course and Complications** The course of the disease is frequently interrupted by febrile exacerbations Early and adequate treatment may effect arrest and considerable improvement but in some cases despite treatment *the course is progressive and the patient becomes* completely helpless Rheumatoid arthritis is associated with psoriasis in about 1% of cases and occasionally with Sjogren s disease (see also p 16)

**Differential Diagnosis** A distinction is made from osteoarthritis both by the clinical findings and X ray evidence of bony overgrowth in the latter Gout is differentiated by the absence of tophi or other uratic deposits and by normal blood uric acid findings Gonococcal arthritis is sometimes mistaken for rheumatoid arthritis The favourable response of the former to penicillin differentiates

**Prognosis** With adequate and early treatment about 15% of patients may be cured and a further 35% improved About 50% of patients remain stationary or get worse despite all forms of treatment

**Treatment** As in pulmonary tuberculosis the patient must be prepared for a long illness and the cardinal principles of treatment are the same rest (general and local) fresh air and an adequate well balanced dietary A search should first be made for a focus of infection The gums nose and throat should be examined and the teeth and sinuses X rayed Bronchiectasis is sometimes overlooked If infection is found at any of these sites it should be suitably treated The patient should rest in bed for a preliminary period of 2 to 3 months or longer *The knees must be straight and not flexed over a pillow* The temperature and pulse rate should be recorded a blood count made and the sedimentation rate of the red cells and the Wassermann reaction determined Iron (Tab Fersolate gr 3 2 t i d ) should be given if there is anæmia and in some cases considerable improvement is effected by a blood transfusion of 500 ml or by the intravenous injection of iron After about 3 months the patient should be allowed gradually to get up *provided the temperature is normal and no fresh lesions are appearing* Local treatment during the acute stage implies rest to the affected joints which are splinted in the optimum position The splints should

collagen diseases in which the collagen substance of connective tissue is affected The small joints of the hands are especially involved There is periarticular swelling, and in the early stages excess of fluid in the joints hyperæmia, lymphocytic infiltration of the joint tissues, and later overgrowth of the synovial membrane with villous projections Decalcification of bone may be followed by destruction of cartilage and later dislocation with bony ankylosis may ensue A rapid atrophy of the small muscles of the hands, and of the larger muscles such as the extensors of the knee and wrist may occur when the fingers knees or wrists are affected Fibrositic changes are also common in the neighbouring muscles Subcutaneous nodules occur in about 10% of cases These consist of an area of central necrosis surrounded by large mononuclear cells and an envelope of fibrous tissue

**Clinical Findings** It should be realised that the disease is a general one with local joint manifestations There is usually a prodromal period of fatigue often accompanied by loss of weight The patient then notices pain and stiffness especially on waking in the morning around a metacarpo phalangeal or interphalangeal joint This is followed by increased pain and swelling of the affected joint or joints The hands and feet are often cold Later the wrists elbows knees ankles cervical spine shoulders temporo mandibular or other joints are affected A periodicity of a few weeks is noticed in some cases between the appearances of fresh lesions During the early stages acute exacerbations may occur from time to time characterised by a throbbing pain disturbing sleep The pain begins suddenly and the patient finds a small red thickening in the skin at the site of the pain usually over an interphalangeal joint The patient is very much disabled if the wrists and hands are affected and cannot exert any power in carrying pulling or gripping partly owing to pain but chiefly because of muscle weakness When the disease is widespread the nights are usually worse than the days The patient often cannot sleep for more than 1 to 2 hours at a time and is then awakened by the pain frequently in the shoulders or knees Movement to a more comfortable position produces pain in many joints The patient cannot lie on either shoulder if they are involved and sleeps best semi propped up On waking in the morning there is much generalised muscle stiffness which takes some time to wear off

*On Examination* In the early stages a spindle shaped deformity is seen usually of the proximal interphalangeal joints with some swelling over the knuckles Small fibrous nodules may be felt subcutaneously on the palmar or lateral aspects of the phalanges Subcutaneous nodules may also be felt especially on the dorsum of the forearm Later if adequate precautions have not been taken much deformity may develop in the hands with ulnar deviation at the wrist and metacarpophalangeal joints and in advanced cases inability to move or use the fingers or thumbs The tissues around the wrists or other large joints, such as the ankles and elbows may be thickened and pit on pressure with limitation of movement There is usually marked wasting of adjacent muscles as described above The skin of the fingers becomes

be removed daily and the patient should put the joints through their full range of comparatively painless movement, but no force should be used, otherwise increased pain and swelling will result Later, various local treatments may be ordered such as active and passive movements gentle massage radiant heat paraffin wax or contrast baths The wax used should have a melting point of 118° to 120° F, and the temperature of the bath should be about 115° F The hands or feet may be plunged into the wax and kept there for 15 minutes or the wax may be painted over them The contrast baths consist of putting the hands alternatively in water at 105° to 110 F for 1½ minutes and then in cold water at 60° F for ½ minute for 20 to 30 minutes Aspirin is the most valuable drug for the relief of pain and should be given regularly during the acute and sub acute stages It appears to have a curative as well as an analgesic action, and if the pain is unrelieved tab codein co may also be given once or twice a day The latter usually produces constipation The usual dose of aspirin required is gr 10 4 or 6 times daily Opinions differ as regards the advisability of using gold salts Good results are claimed in about 50% of cases treated in Great Britain and in about 35% in America The treatment is certainly dangerous and its risks should be explained to the patient before it is initiated A preparation such as inject sod aurothiomalat (B P) mg 10 in 1 ml is injected intra muscularly at weekly intervals beginning with mg 10 and increasing to mg 20 and for the third and subsequent doses mg 50 until a total of 0 75 to 1 G has been injected A second and third course may be given after a rest period of 3 months Severe and fatal reactions may occur, especially exfoliative dermatitis agranulocytosis and purpura At the slightest sign of a skin eruption no more gold should be injected Renal and hepatic disease and severe debility are contra indications Gold should only be given if the sedimentation rate of the red cells is over 25 mm (Westergren) at 1 hour the urine free from albumin and the white cell count normal A complete blood count and urine examination should be made weekly and a sedimentation test monthly Gold should not be continued if the sedimentation rate is less than 25 mm (Westergren) at 1 hour If the eosinophil count rises over 5% the next dose of gold should be halved If there is albuminuria no further injections should be given Unless these precautions can be taken gold should not be used If symptoms of gold poisoning occur B A L should be injected (see p 546) Cortisone was originally obtained from adrenal cortex It was then synthesised from bile salts and now is prepared from "elephant s foot" a root which grows in South Africa and also from the juice of sisal which is grown in East Africa ACTH is the pituitary adrenocorticotropic hormone, which stimulates the production of cortisone Both these substances are used for the treatment of rheumatoid arthritis They do not cure the disease but effect a rapid improvement in the symptoms with increased freedom of joint movement diminution of pain, increased muscular strength disappearance of fever fall in the sedimentation rate of the red cells and they also produce a general sense of well being accompanied by refreshing sleep Certain unfavourable side effects may be produced

These include retention of sodium with œdema symptoms of Cushing s syndrome roundness of the face and hirsuties acne osteoporosis mental excitation and psychotic states These drugs should not be given if the patient has an active infection in any part of the body or if a tuberculous focus is present in the lungs or elsewhere Other contra indications include cardiac failure renal disease, osteoporosis, peptic ulcer and mental instability Hyperglycæmia may also occur and if these drugs are given to a patient suffering from diabetes mellitus and rheumatoid arthritis special care is required in controlling the diabetes In some cases there is excessive excretion of potassium which results in muscular weakness and in electrocardiographic changes (see p 505) Cortisone is usually preferred to ACTH for ACTH is rapidly used up and has to be given by intramuscular injection usually every 6 hours The initial dose of ACTH is mg 12 5 In some cases mg 25 every 6 hours are required to produce a favourable response

Cortisone is usually given by mouth The initial dose is mg 25 three or four times a day Subsequently the dose is slowly reduced until the correct maintenance dose is determined It is usually necessary to continue the cortisone indefinitely a relapse occurring within a few days of leaving off the maintenance dose Hydrocortisone is used for injection into a joint such as the knee, in doses of mg 25 to 50 usually at weekly intervals Prednisone and prednisolone are analogues of cortisone and hydrocortisone They are about four times as potent as cortisone and so the initial dose is usually mg 5 three or four times daily a maintenance dose of about mg 7½ to 10 in divided doses being often sufficient There is little to choose between these two prepara tions They have the advantage of not causing retention of sodium or loss of potassium to any degree On the other hand gastric upset is very likely to occur peptic ulcer may form and there may be hæmatemesis A moon shaped face also is seen as in cortisone treatment Butazolidin is dangerous unless given in small doses as it is liable to cause œdema anæmia or agranulocytosis The dose is 1 tab mg 100 t i d for two or three days A maintenance dose of mg 100 to 150 daily may usually be safely given Contra indications to Butazolidin are cardiac damage hypertension renal or hepatic lesions a history of peptic ulceration of blood dyscrasias or of drug allergy Orthopædic treatment may be required to correct deformities in cases inadequately treated during the early stages The patient should not be allowed to return to work until arrest has been secured as judged by a normal temperature and sedimentation rate a normal body weight ab ence of swelling and stiffness and comparative freedom from pain in the affected joints

## Disseminated Lupus Erythematosus

The characteristic features of this disease are fever polyarthritis polyserositis endocarditis erythematous cutaneous lesions nephritis anæmia a remittent cachexial course with usually a fatal termination months to years after the onset All these features are not necessarily present in each case It frequently affects young women 95% of cases

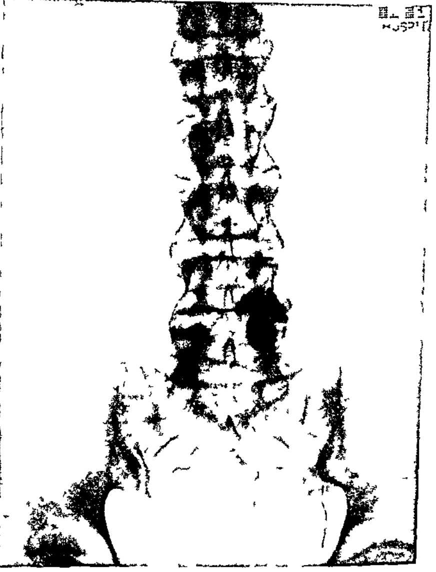


Fig 62 The Bamboo Spine in Rheumatoid Spondylitis

rheumatic fever The sedimentation rate of the red cells is rarely increased The B M R is below normal in about 30% of cases Radiographic examination reveals cyst like structures in the bones on the joint surface which result in erosion of the joint cartilage with secondary osteophytic outgrowths

**Course and Complications** Several joints may be affected, and one or more of them may be completely immobilised The smaller joints of the fingers may be involved especially in middle aged women, with development of Heberden s nodes at the proximal ends of the distal phalanges At the climacteric osteoarthritis may occur in the knees, associated with obesity

**Differential Diagnosis** This is as for rheumatoid arthritis Limitation of external rotation and abduction of the hip joint distinguishes a case of osteoarthritis of the hip from sciatica Acute osteoarthritis can usually be distinguished from acute rheumatic fever by the failure of response to salicylates

**Prognosis** This is unfavourable as regards recovery, but the disease does not progress to a stage of complete crippledom

**Treatment** Massage and radiant heat increase the vascularity of *the structures around the joint and help to relieve pain* The joint should be rested as much as possible the patient lying down if the knees or hips are affected for at least an hour daily No strain should be put on the affected joints but they should be put through their full range of movement two or three times a day Exercises which produce discomfort lasting for more than 2 hours are excessive If the patient is obese and the knees and hips are involved dietetic restrictions should be imposed Thyroideum gr ½ to 1 t d s is of value in cases associated with obesity or the climacteric Gold salts are of no value Physical treatment such as deep X rays diathermy and paraffin wax baths may afford considerable relief from pain Hydrocortisone may be injected into the knee joint with relief of symptoms In addition if only one joint such as the hip is involved operative measures may be considered such as moving the joint under an anæsthetic osteotomy of the femur to correct deformity *removal of foreign bodies* from the joints excision of the bony outgrowths which are preventing movement (cheilectomy) vitallium cup arthroplasty and arthrodesis Surgical appliances such as Thomas walking caliper will take the weight of the body from the pelvis off the hip

## Rheumatoid Spondylitis

(*Atrophic Spondylitis Spondylitis Ankylopoietica Von Bechterews Disease Strumpell Marie Disease Spondylose Rhizomélique*)

**Definition** A rheumatoid type of arthritis affecting the spine

**Etiology** This is uncertain but it is usually considered to be the same as for rheumatoid arthritis *Predisposing causes* 1 Age 20 to 30 years 2 Sex Males predominate

**Pathology** There is synovitis of the posterior intervertebral joints with osteoporosis of the vertebral bodies The sacro iliac and

being females When endocarditis is a prominent feature the disease is known as the Libman Sacks syndrome No organisms are present in the verrucous endocarditis the vegetations tending to spread over the valve surfaces and mural endocardium Polyserositic manifestations are prominent in some cases with ascites and pleural effusion Such cases usually develop glomerulonephritis The skin lesions assume a butterfly distribution on the face and small red spots come and go on various parts of the body such as the fingers, elbows and on the scalp The primary lesion appears to lie in the collagen tissue which serves as a matrix for the capillaries The L E cells of Hargreaves are of diagnostic value They may be found in the blood or bone marrow They are phagocytes usually neutrophil polymorphonuclears containing masses of chromatin which take a deep purple colour with Giemsa s stain

Cortisone or ACTH produces a remission of the symptoms but in severe cases death usually occurs despite continuance of the treatment The appearance of albumin red cells and casts in the urine may be the first indication of the true nature of the disease

## Osteoarthritis

(*Hypertrophic Arthritis Degenerative Arthritis*)

**Definition** Degenerative joint lesions of doubtful etiology

**Etiology** Trauma and strain are exciting factors in some cases *Predisposing causes* 1 Age Usually over 40 2 Sex Males predominate 3 Metabolic errors The nature of these is uncertain 4 Obesity

**Pathology** The joints most commonly affected are the terminal interphalangeal joints where Heberden s nodes occur the lumbar vertebræ the knees the sacro iliac joints the lower cervical vertebræ, the hips and shoulders There is degeneration of the articular cartilage with hypertrophy of the surrounding cartilage and bone so that the articular surfaces are approximated The synoviæ are usually unaffected Separated osteophytes may be loose in the joints The joint capsule is fibrosed Osteoarthritic changes are present in nearly everyone after the age of 50 but symptoms only occur in a small percentage of individuals

**Clinical Findings** The onset is usually insidious with stiffness and later pain in the affected joints There is no constitutional disturbance except in the rare acute type of disease When the hip is affected the pain may be referred to the front or back of the leg (crural or sciatica neuralgia) or to the knee

*On Examination* The general nutrition is good The affected joint shows some bony thickening and lipping of the bony edges may be felt Creaking is often audible and palpable on movement, and the range of movement is limited Some muscular wasting may occur above or below the joint, and in cases in which the hip joint is affected the patient may walk on his toes with tilting of the pelvis and scoliosis In some instances the disease has an acute onset closely resembling that of acute

### Still's Disease

**Definition** A variety of rheumatoid arthritis occurring in children and characterised by swelling of joints enlargement of the spleen and lymph glands with pyrexia A similar condition may affect adults when it is known as Felty s syndrome

**Clinical Findings** The disease usually begins before the second dentition The parents notice swelling in the joints usually in the hands knees or wrists and the child complains of pain and stiffness in them

*On Examination* The joints show periarticular swelling the finger joints may be spindle-shaped and movement of the joints is painful and limited The wrists knees and cervical vertebræ may also be affected Enlarged glands may be felt generally distributed and the spleen may be palpable The muscles near the affected joints atrophy the child is pale sweats and runs an irregular temperature of about 100 F

**Course and Complications** In the majority of cases the disease pursues an irregular course with remissions and exacerbations and proceeds to a fatal issue from some intercurrent disease Internal adherent pericardium may occur

**Prognosis** This is grave but recovery has been recorded

**Treatment** The patient must be kept in bed during the active stages of the disease and splints applied to prevent deformities If the tonsils are infected they should be removed The effect of cortisone treatment remains to be seen

### Specific Infective or Toxic Arthritis

The inflammation of joints is here due to infection with known organisms or occurs as a complication of infective diseases This group includes pneumococcal tuberculous gonococcal staphylococcal and streptococcal arthritis acute rheumatic fever and arthritis associated with syphilis dysentery (bacillary) Malta fever dengue typhoid fever cerebro-spinal fever scarlet fever measles and mumps

Other varieties of arthritis are as follows Metabolic as in gout Hæmorrhagic as in purpura and hæmophilia Nervous as in Charcot s joints or in syringomyelia Anaphylactic as in serum disease Associated with deficiency diseases such as rickets or scurvy Psoriatic arthritis This may be rheumatoid arthritis complicated by psoriasis It is said that the terminal interphalangeal joints are especially liable to be affected This is not always so Traumatic and intermittent hydrarthrosis Here periodical swelling occurs in joints especially the knees and wrists There is considerable weakness but little pain The swelling usually subsides in 2 to 3 weeks It is possibly allied to angio neurotic œdema

Treatment consists in rest and firm bandages

### Reiter s Disease

This syndrome consists of polyarthritis non specific urethritis and conjunctivitis There may be fever and diarrhœa with blood in the stools at the onset By some it is thought to be due to bacillary dysen tery

costovertebral joints are often affected Calcification occurs in the ligaments of the spine, and in the lateral borders of the intervertebral discs The spine thus becomes rigid the bamboo spine

**Clinical Findings** The onset is usually insidious with pain and stiffness in the back especially on waking in the morning Root pains may occur in the arms or legs or girdle pains around the chest or abdomen In a few cases there is an acute onset with fever

*On Examination* There is rigidity of the back (poker back), the spine may be curved with kyphosis in the upper thoracic region In the early stages there is considerable spasm of the erector spinæ muscles Flaccid paresis of the legs may occur from root pressure with paræsthesia or cord compression may give rise to spastic paralyses There may be acute pain on percussion over the lateral spinous processes Later the patient may be unable to stand upright to turn without moving the whole body or to take a deep breath There are no rheumatic nodules The sedimentation rate of the red cells is increased X ray examination shows decalcification of the bodies of the vertebræ the joint spaces are not usually narrowed until late in the disease The spinal ligaments are calcified (see Fig 62 p 641)

**Differential Diagnosis** The diagnosis is established by the X ray examination of the spine Changes are first seen in the sacro iliac joints If there are no bony changes the stiffness is usually due to fibrositis of which chronic gonorrhœa may be the cause A spinal tumour or syphilitic meningitis may cause similar symptoms but there are no bony changes The pain may suggest biliary or renal disease scoliosis pleurisy, Pott s disease or sacro iliac strain

**Course and Complications** The course is usually slowly progressive Respiratory complications such as bronchitis render the outlook more unfavourable Pulmonary tuberculosis occurs in about 25% of cases The hips shoulders temporo mandibular sterno clavicular and manubrio sternal joints may all be affected

**Treatment** The general treatment resembles that advised for rheumatoid arthritis A spinal brace should be worn to relieve the strain on the back X ray treatment is particularly valuable in the early stages for relief of pain increasing mobility and preventing deformity

## Osteoarthritic Spondylitis

*(Hypertrophic Spondylitis)*

**Pathology** Degenerative changes occur in the spine similar to those described under osteoarthritis

**Clinical Findings** Although osteoarthritic changes are present in the spine in the majority of people over the age of 50, they rarely give rise to symptoms The chief symptoms are root pains some rigidity of the spine and muscular atrophy When the cervical spine is involved there may be pain in the shoulders neck and arms with limited spinal movement and Horner s syndrome

**Treatment** This includes rest to the spine heat massage and in some cases a spinal support

i u and an infant 1 500 i u daily Cod liver oil 280 drops or halibut liver oil 10 drops contains 700 i u Absence of this vitamin from the food of puppies is followed by rickets In human beings deficiency of this vitamin may cause rickets or dental caries and overdosage results in irritability anorexia diarrhœa excess of calcium in the bones and deposition of calcium in the kidneys Success has attended the administration of large doses of vitamin D in the treatment of lupus vulgaris 100 000 to 150 000 i u being given daily either as a 50 000 i u tablet t i d or as a capsule containing 100 000 i u The treatment is prolonged for several months If the patient complains of nausea or vomiting the dose should be reduced The blood calcium should be estimated at the end of 6 weeks

*Vitamin E* (Fertility or anti sterility Vitamin ) This fat soluble vitamin is present in olive oil wheat embryo and green leaves Wheat germ oil is its most potent source Four substances $\alpha$ $\beta$ $\gamma$ and $\delta$ tocopherol have been isolated each possessing vitamin E activity There is no proof that vitamin E is of any value in the treatment of habitual or threatened abortion or of the myopathies It is essential for reproduction in rats

*The Vitamin B Complex* This vitamin has been divided into several factors The following constituents have been isolated and synthesised aneurin riboflavin nicotinic acid pyridoxin biotin folic acid p amino benzoic acid and panthothenic acid It occurs in seeds eggs the germ and bran of cereals including the husk of rice and in yeast meat fish milk etc Its presence in bread varies with the amount of wheat germ used

*Vitamin $B_1$* This water soluble vitamin has been isolated in pure form and also synthesised The hydrochloride is known as aneurin or thiamine An adult requires about 700 i u daily and 3 mg of synthetic vitamin $B_1$ hydrochloride are equivalent to 1 000 i u Deficiency leads to beri beri and possibly to certain types of peripheral neuritis in man and to polyneuritis in pigeons It is believed that minor degrees of deficiency will cause in animals and man anorexia constipation digestive disturbances and liability to intestinal intoxication Pink disease in children may be due to lack of vitamin $B_1$

*Vitamin $B_2$* This contains riboflavin (lactoflavin) and nicotinic acid *Riboflavin or Lactoflavin* Lack of this vitamin may be responsible for the glossitis and angular stomatitis met with in pellagra Rosacea keratitis also responds to its administration

*Nicotinic acid or Niacin* (Vitamin P P or pellagra preventing) In addition to its use in the prevention and treatment of pellagra nicotinic acid is also used on account of its vasodilator action in the treatment of cerebral thrombosis angina pectoris and intermittent claudication

*Vitamin $B_6$* (Pyridoxin) This is the anti dermatitis factor in rats In dogs and pigs its lack results in hypochromic microcytic anæmia

*Biotin* Deficiency in rats causes a scaly dermatitis

*Folic acid* This is found in green leaves spinach tops yeast and liver It is concerned with erythropoiesis and used in the treatment of macrocytic anæmia

# CHAPTER XI

# DISORDERS OF METABOLISM

## DEFICIENCY DISEASES

**Introductory** Certain disorders are believed to be due to a deficiency or absence of a vitamin from the food They are Xerophthalmia night blindness rickets beri beri rosacea keratitis pellagra scurvy, bleeding associated with jaundice and neonatal hæmorrhage Vitamins or accessory food factors are substances present in food which are essential for growth or health Only minute quantities are necessary They fall into two groups according to their solubility in fats or water The following are described *Vitamin A* (Growth Vitamin, anti infective anti xerophthalmic) This is derived from green leaves where it is probably synthesised with the aid of light It can also be formed apart from the action of light It is present in animal fats in milk butter cod liver and halibut liver oil etc as the result of food eaten by the animal It is found in traces only in vegetable oils Carotene ($C_{40}H_{56}$) which is present in vegetables is its precursor or the pro vitamin Some authorities consider that carotene is con verted into vitamin A in the liver and stored there The vitamin A content of milk is lowest in January and highest in June Vitamin A ($C_{22}H_{32}O$) is destroyed by sunlight or by heating in air It gives a blue colour with arsenic or antimony trichloride The 'blue unit' or Carr Price unit is based on this colour change The international unit of 1949 is 0 34 microgramme of a standard preparation of vitamin $A_1$ acetate representing 0 3 microgramme of vitamin $A_1$ alcohol An adult requires 3 000 i u and a child 1 500 to 3 000 according to age daily During pregnancy and lactation a woman requires 6 000 i u A teaspoonful of cod liver oil contains 6 400 i u and a drop of halibut liver oil 3 200 i u Absence of this vitamin from the food of young animals arrests their growth Deficiency of vitamin A in man may cause dryness of the skin and hyperkeratosis, night blindness and keratinisation of the epithelium in the eyes lungs genito urinary tract and mouth with a liability to secondary infections Excess of the vitamin is harmless to man

*Vitamin D* (Anti rachitic Vitamin) This is present in animal fats such as cod liver and halibut liver oil but butter is poor in it *It is* present in vegetable oils It is not destroyed by heat and probably not by sunlight It has a characteristic ultra violet absorption spectrum Calciferol can be synthesised from ergosterol an impurity of cholesterol by ultra violet rays Ergosterol is regarded as a pro vitamin D there are at least 9 others known Calciferol (vitamin $D_2$) prepared from ergo sterol possesses a very high antirachitic activity *i e* 40 000 i u per mg, and is closely related to the naturally occurring vitamin D The international unit of vitamin D is the activity of 0 025 microgramme of calciferol (irradiated ergosterol or vitamin $D_2$) An adult requires 150

and orange peel It is available for clinical use as Hesperidin in the treatment of certain cases of petechial hæmorrhage

## Rickets

**Definition** A general metabolic disorder of infants with characteristic *changes in the bones especially near the epiphyses*

**Etiology** Rickets may be caused by 1 Errors of diet, especially deficiency of vitamin D and calcium Excess of cereals produces an antagonistic effect on vitamin D 2 Absence of sunlight The ultra violet rays are antirachitic and perhaps effect a synthesis of the vitamin D They do not pass through ordinary glass 3 Defective hygiene and confinement *Predisposing causes* 1 Age 6 to 18 months 2 Season Winter and spring 3 Climate and locality In the temperate zones and in large cities 4 Absence of breast feeding Rickets however often does develop in a breast fed baby 5 Prolonged breast feeding After 1 year 6 Rapid growth

**Pathology** *The Bones* The calcium phosphate content is low about 21% instead of the normal 63% and owing to lack of calcium the bones bend Changes are seen in the long bones near the epiphyses *the end of the bone is enlarged and the epiphyseal line is widened* beyond the normal 2 mm and uneven owing to irregular and excessive proliferation of the cartilage cells Normal ossification does not ensue and the marrow is unduly vascular The deficiency of calcium and phosphorus in the bone is probably due to failure of absorption from the intestine The blood phosphorus and calcium are usually low in active rickets such as Ca 6 and P 2 mg per 100 ml (normal figures Ca 9 to 11 and P 2 5 to 5 mg per 100 ml) It is believed that calcium phosphate is normally deposited in growing bone owing to the action of an enzyme phosphatase formed in bone Phosphatase also occurs in the intestinal mucous membrane in the kidneys and in the blood The normal plasma alkaline phosphatase figure is 3 to 13 units (King and Armstrong method) In rickets the plasma phosphatase is increased during the active stages of the disease The liver may be *enlarged and fatty and the spleen fibrous As the condition improves* the calcium and phosphorus contents of the blood rise and the phosphatase content falls

**Clinical Findings** The patient is usually an infant aged about a year a history of improper feeding may or may not be obtained the symptoms are generally noted in the winter or spring they include irritability restlessness and sweating on the head at night the child may also suffer from diarrhœa bronchitis convulsions tetany or laryngismus stridulus in many cases he is not brought for treatment until the disease is more advanced and advice is sought on account of delay in walking in speaking or in dentition or for curving of the limbs

*On Examination* The child is usually pale but well covered The earliest sign of rickets is craniotabes which can be detected in a premature baby at the age of 1 to 3 months *Softened areas of bone* are felt especially behind the ears A month or so later enlargement

*Vitamin* $B_{12}$ This was isolated from the liver in 1948 mg 20 of the vitamin being obtained from 1 ton of liver For further information see pp 436 516

Vitamin B nutritional deficiencies were met with during the 1939–45 war amongst the troops serving in the East Florid deficiency states were rare but subclinical deficiencies were more common Most of the food in forward areas was from tins There was little fresh food in the battle zone in static phases, and almost none on advance or withdrawal Synthetic vitamins were difficult to obtain Symptoms of beri beri were rare, and when found were often associated with some other disease Pellagra was exceptional Corneal vascularisation requires for its detection slit lamp examination, which was often not available and as a test for riboflavin deficiency was not found to be of much value Nutritional neuropathy is an interesting condition which occurs in pellagra and beri beri Its main features are burning feet, retrobulbar neuritis and scotomata nerve deafness laryngeal palsy and more rarely ataxia due to pyramidal or posterior column lesions In some cases the blood pressure is raised In prison camps in the Far East it was found that nicotinamide was the most useful single vitamin for relieving burning feet but the neuropathy was generally treated with any food available rich in vitamin $B_2$ frequently one of the pulses growing in wild form Crystalline $B_1$ given parenterally had no effect on the nervous lesions and the severe cases of optic nerve involvement or ataxia, often failed to respond to treatment Pellagra, glossitis scrotal eczema cheilosis, and diarrhœa often fatty were rife in most Japanese prison camps from time to time Some cases of Wernicke's encephalopathy were also described with vomiting and nystagmus as early symptoms, ophthalmoplegia and mental disturbances occurring later

*Vitamin C* (Anti scorbutic Vitamin) The following are particularly good sources of vitamin C —Hips haws blackcurrants and Brussels sprouts Sprouts should be cooked by cutting them up and dropping them into boiling water taking care the water does not go off the boil ' The vitamin is not then likely to be destroyed by oxidases during cooking It is now believed that ascorbic acid is the vitamin It is destroyed by heat in the presence of air if the reaction is alkaline It can be prepared in bulk from certain peppers of the genus capsicum It is generally absent from tinned fruit but it is present in fruit canned anaerobically as heat does not destroy it in the absence of oxygen Adults require 50 to 100 mg of ascorbic acid (1 000 to 2 000 i u ), and infants 15 to 50 mg of ascorbic acid daily Its deficiency causes scurvy in infants and in adults and possibly faulty enamel formation in teeth

*Vitamin K* This is widely distributed amongst plants and a synthetic substance menaphthone 2 methyl 1 4 naphthoquinone is available for clinical use as Menaphthonum B P Its deficiency causes a diminution of prothrombin in the blood It is valuable in checking hæmorrhage associated with jaundice and neonatal hæmorrhage There is deficient absorption of vitamin K when bile is absent from the intestine

*Vitamin P* This is present in Hungarian red pepper, lemon juice

baby cannot take cod liver oil he should have general ultra violet light radiation every other day One and a half pints of milk should be given daily during the second year of life

*Curative* The child should be taken off his feet during the acute stage and treated with 5 000 i u of vitamin D daily (= m 1 000 of cod liver oil or 72 drops of halibut liver oil) Concentrated preparations of irradiated ergosterol are available such as Haliverol and calciferol Haliverol (halibut liver oil with added vitamin D) contains 250 times as much vitamin D as does cod liver oil Calciferol is put up as liq calciferol (B P ) m 5 = 1 000 i u Ultra violet light or irradiated milk 12 to 18 fl oz daily with an equal amount of unirradiated milk for a child of 2 to 5 is helpful in some cases Overdosage (hyper vitaminosis) must be avoided as the bones may become prematurely ossified and calcium be deposited in the kidneys If there is marked deformity of the limbs splints may be required this also keeps the child off his feet The limbs should be massaged X rays show evidence of healing first at the end of the ulna radius or femur as a new calcification zone between the epiphysis and diaphysis Diet Excess of starchy foods must be avoided The diet should consist of milk $1\frac{1}{2}$ pints rusks made from wholemeal bread yolk of an egg orange juice and a little porridge gravy greens and steamed fish

## Infantile Scurvy

(*Barlow s Disease*)

**Definition** A disease characterised by hæmorrhages into the skin mucous membranes and under the periosteum without characteristic blood changes

**Etiology** Infantile scurvy is due to a deficiency of vitamin C in the diet It is a disease of infancy manifesting itself between the ages of 8 and 12 months It develops in infants fed on boiled pasteurised dried or condensed milk who have not been supplied with the vitamin as should be done by giving fruit juice

**Pathology** There is increased capillary permeability At autopsy hæmorrhages may be found under the periosteum especially of the long bones the epiphyses may be separated or the bones fractured There is inhibition of the growth of osteoblastic bone and the cartilage is less vascular than normal There may also be infarcts in the lungs and hæmorrhages into the intestines or kidneys

**Incubation Period** The disease manifests itself after deprivation of the vitamin for 6 to 8 months

**Clinical Findings** The onset is usually gradual and the baby may be brought to a doctor on account of screaming when the limbs are touched or because he does not move an arm or leg or for pallor bruising on the face near the orbit prominence of an eye or bleeding from the gums bowel or urinary tract A history of improper feeding is usually obtainable

*On Examination* The infant may be pale wasted and fretful crying when touched Bruising may be seen on the face or body, or petechiæ

of the lower end of the radius, ulna and femur and of the upper end of the tibia may be noted, with beading of the costo chondral junction A transverse depression running round the front of the lower part of the chest called Harrison s sulcus, is seen at the level of the diaphragmatic attachment

Prominence of the sternum may cause a pigeon chest appearance Bossing of the skull is due to prominence of the frontal and parietal eminences (hot cross bun appearance) there is delay in closure of the anterior fontanelle (normally closed in 18 months) The legs arms or spine may be curved and the pelvis flat green stick fractures may occur in the extremities The muscles are weak and the ligaments often lax The liver and spleen are palpable due either to displacement secondary to chest deformity or to enlargement The abdomen is distended Rhonchi may be heard in the lungs The blood usually shows anæmia and the phosphorus and calcium are low and the phosphatase is raised as mentioned above X ray examination The earliest bone changes are the disappearance of the sharp margin of the metaphysis at the lower end of the radius ulna or femur, later the diaphyseal end becomes cupped and the epiphyseal line is widened

*Varieties* Adolescent Rickets (Rachitis Tarda) rickety manifestations appear at puberty, affecting only the long bones The disease is associated with conditions of great privation Adult rickets is exemplified by osteomalacia

**Differential Diagnosis** Congenital syphilis may be suggested by the appearance of the skull and the curving of the bones, the Wassermann reaction of the blood differentiates The enlarged head must be distinguished from that due to hydrocephalus the curved spine from that produced by caries and the muscular weakness from that caused by poliomyelitis or scurvy

**Course and Complications** The course depends largely on the treatment if untreated there is a tendency to cure after the second year of life but bony deformities persist Complications include Bronchitis bronchopneumonia laryngismus stridulus and possibly tetany Permanent deformities may result if treatment is delayed or inadequate with dwarfism and difficulty in childbirth

**Prognosis** This is very good provided the rickets is diagnosed before an advanced stage is reached and that it is treated efficiently Death may occur from convulsions or from laryngismus stridulus Rickets increases the gravity of measles and bronchopneumonia in infants

**Treatment** *Prophylactic* Expectant mothers should have a correctly balanced dietary Every baby at the age of three weeks should be given vitamin D, 700 i u daily such as cod liver oil m 140 or halibut liver oil 10 drops Premature babies require double the amount The dose should be gradually increased to 1 500 i u daily and thus maintained for several years Raw yolk of egg is also a good preventive One half may be given daily for the first 2 months and the whole yolk daily after this The baby should be out of doors every day, and the arms and legs exposed to the sun In winter if the

Seeds such as barley, peas beans or lentils acquire antiscorbutic powers if soaked in water for 24 hours and kept moist for 3 days They then germinate and the sprouts are antiscorbutic Soda, used in cooking green vegetables to preserve their colour destroys their antiscorbutic properties

**Clinical Findings** The sufferer complains of general weakness shortness of breath and vague limb pains Hæmorrhages are then noted from the gums or nose and there may be night blindness

*On Examination* The patient is sallow the gums may form fun gating masses obscuring the teeth which become loose the breath is foul tender lumps due to hæmorrhages may be felt in the skeletal muscles and hæmorrhages are seen in the skin Ascorbic acid is usually absent from the urine

**Treatment** This consists in supplying antiscorbutic substances as described above or by the injection of ascorbic acid 0 5 to 1 G intramuscularly t i d followed by mg 500 tab ascorbic acid by mouth b i d Hydrogen peroxide should be applied to the gums

## Beri beri

### (*Polyneuritis Endemica*)

**Definition.** A deficiency disease characterised by polyneuritis cardiac symptoms and œdema

**Etiology** Beri beri is due to deficiency of vitamin $B_1$ in the diet There is often in addition a lack of nicotinic acid and riboflavin It is believed that thiamine (vitamin $B_1$) is phosphorylated to form a co enzyme co carboxylase This is necessary for the metabolism of pyruvic acid which is derived from the breakdown of carbohydrates If pyruvic acid accumulates the tissue and nerve cells are deprived of the energy derived from the oxidation of glucose The disease occurs in countries where the chief article of food is polished rice deprived of the pericarp which contains the vitamin It is thus endemic in the Malay States Japan and the East Indies It may also occur in prisoner of war camps if the diet is deficient in vitamin $B_1$

**Pathology** At autopsy the heart weighs more than normal there is enlargement chiefly of the right ventricle Serous effusions occur in the pericardium and pleuræ The liver and spleen are congested and the lungs œdematous The mucous membrane of the stomach and duodenum may be congested The nervous system There may be degeneration of anterior horn cells in the cord and of posterior ganglion cells In the peripheral nerves the myelin sheath degenerates and the axis cylinder may rupture the sympathetic fibres may also degenerate and vagus and phrenic neuritis may occur The heart failure is possibly due to water retention in the cardiac muscle rather than to vagal neuritis

**Incubation Period** Symptoms usually appear after deprivation of vitamin $B_1$ for 3 months

**Clinical Findings** There are two main types of the disease dry and wet The patient is usually a young adult but infants of mothers suffering from beri beri may show symptoms of the disease

on the palate Hæmorrhages are only seen from the gums in the neighbourhood of erupted teeth An arm or leg may appear paralysed the child not moving the limb owing to pain A tender swelling may be felt deep to the muscles often towards the lower end of the femur There may be œdema of the face or limbs and the urine may contain blood or a trace of albumin There may be proptosis of an eye, due to retro orbital hæmorrhage The temperature is usually a little raised about 100° F and the pulse frequent The blood This shows an anæmia but the platelet count, bleeding time and coagulability are normal The plasma alkaline phosphatase is low X ray examination may reveal a subperiosteal swelling separated epiphysis or fracture Fraenkel s " white line " is a dense transverse shadow at the junction of the epiphysis and diaphysis

**Differential Diagnosis** The following conditions should be considered —Purpura or leukæmia The gums are not affected in purpura and there is no dietetic error In leukæmia the blood count is diagnostic Osteomyelitis and poliomyelitis The presence of hæmorrhages are characteristic of scurvy the X ray appearances also help to differentiate osteomyelitis there is no real paralysis present and the deep reflexes are unaltered Acute rheumatism does not occur below the age of 2 years Syphilitic epiphysitis is usually seen under the age of 3 months Trauma may be suggested by the bruising or a retro orbital growth if there is proptosis

**Course and Complications** Adequate treatment rapidly checks the course of the disease Complications include —Bronchopneumonia, otitis media furunculosis nasal diphtheria and diarrhœa Rickets may also be present (scurvy rickets)

**Prognosis** The infant should be cured in a week with proper treatment

**Treatment** *Prophylactic* Vitamin C must be supplied from birth in the form of orange blackcurrant or rose hip juice sweetened and diluted with water The protective daily dose for an infant is ascorbic acid mg 15 to 50 50 mg of ascorbic acid is contained in 3 fl oz of fresh orange juice 1 fl oz of blackcurrant juice, or $\frac{1}{3}$ fl oz of rose hip juice

*Curative* Orange blackcurrant or rose hip juice should be given daily as above and in addition ascorbic acid tab mg 100 should be given t i d While the limb is painful the baby should be disturbed as little as possible and the limb supported on a splint

## Adult Scurvy

**Etiology** James Lind an officer of the British Navy showed in 1753 that scurvy is not an occupational nor an infective disorder but is due to the lack of certain foodstuffs He introduced lemon juice into the seamen s ration Scurvy ravaged Paris during the siege in 1870 and was met with in Russia Rumania and Mesopotamia during the 1914–18 war

The juice of sweet limes or lemons from the Mediterranean is preventive but the sour limes from the West Indies have not this property

*Predisposing causes* 1 Age 20 to 40 years 2 Sex Slightly more common in females 3 Season Chiefly in spring and early autumn 4 Locality Especially Italy Rumania, South East America Egypt and India and in country rather than in town dwellers 5 Debilitating conditions such as poverty recurrent malaria, syphilis tuberculosis and alcoholism

**Pathology** At autopsy the body is wasted and dermatitis may be seen on exposed areas of skin There is atrophy of the muscle and mucous membrane of the stomach and intestines, and ulcers may be found in the colon The heart is small The liver may be fatty or cirrhotic There is degeneration of the posterior and lateral columns of the cord but no peripheral neuritis Meningeal thickening may be present in the brain or cord

**Clinical Findings** The patient is usually an adult who notices in the spring loss of appetite nausea vomiting or diarrhœa He may also complain of soreness of the mouth or tongue, and excessive salivation Later redness and itching of exposed areas of the skin may be noted The patient may also complain of numbness or cramps in the legs and of headache giddiness or insomnia

*On Examination* Affected areas of skin may be seen on the backs of the hands and forearms, on the forehead on a butterfly area of the face and on the dorsum of the feet They are symmetrical resembling sunburn with definite edges They darken and blebs may form during the summer the colour fades and the skin desquamates and after several years the affected skin becomes thin Recurrence may occur in the autumn or not until the next spring The tongue is often red and glazed and angular stomatitis is seen a fractional test meal may reveal achylic gastrica blood or mucus may be seen in the motions A spastic or flaccid paresis may be found in the legs the former being associated with achlorhydria The cerebro spinal fluid is normal but the blood often shows an anæmia The temperature is usually normal An acute variety of the disease may occur but the cases described as pellagra sine pellagra are of doubtful etiology

**Differential Diagnosis** The skin lesions are diagnostic and differentiate pellagra from such diseases as sprue or spastic paralysis due to other causes

**Course and Complications** There is always a tendency to seasonal recurrences of increasing severity wasting and mental deterioration in these cases gradually ensue with permanent paralysis

**Prognosis** Apart from the acute cases which survive for only a few weeks patients often live for 20 years or longer after the diagnosis is made Many cases die in the asylum with delusions or dementia, if adequate treatment has not been instituted early

**Treatment** *Prophylactic* A well balanced diet is essential containing lean meat fruit vegetables eggs milk 3 pints a day and butter

*Curative* During the acute stages the patient should be put to bed and nicotinic acid mg 100 given 5 times a day by mouth or nicotinamide mg 50 to 200 in normal saline injected intravenously

The patient complains of weakness numbness or tingling in the legs, dyspnœa on exertion palpitations tender spots in the calves and swelling of the legs

*On Examination* In the "dry" type signs of peripheral neuritis are found such as weakness and wasting of the leg muscles, areas of cutaneous anæsthesia or hyperæsthesia, depression or loss of the deep reflexes, and the reaction of degeneration is obtained Localised tender swellings may be present in the calf (probably due to water retention in the muscles) The skin is dry In the "wet" variety œdema is present in the legs and free fluid may be found in the abdomen, pleuræ or pericardium The heart is enlarged chiefly to the right the apex beat becomes diffuse and fluttering, the rhythm remains regular and the pulse is soft and unduly frequent The electrocardiogram shows no abnormality beyond right axis deviation The cervical veins are engorged and the liver may enlarge and pulsate Œdema of the lungs is a terminal event In addition neuritis may be present The temperature usually remains normal An acute variety is described in which the patient dies in a day or so from heart failure Rudimentary forms also occur with recurrent pain or weakness in the legs

**Differential Diagnosis** The occurrence of œdema and peripheral neuritis with heart symptoms in a locality in which beri beri is endemic is diagnostic Peripheral neuritis due to other causes must be excluded in the "dry" variety and a careful enquiry made into the diet Vitamin $B_1$ deficiency in the diet can be detected by estimation of its excretion in the urine and by the pyruvate tolerance test

**Course and Complications** The course is usually progressive unless the dietetic error is corrected

**Prognosis** Death may occur suddenly from heart failure Recovery is the rule with adequate treatment

**Treatment** *Prophylactic* An ordinary mixed diet is all that is requisite in rice eating countries some of the husk of the rice should be left after milling Yeast or Marmite contains the vitamin and can be eaten when fresh food is scarce or white flour used in the making of bread may be fortified with synthetic vitamin $B_1$

*Curative* The vitamin $B_1$ can be supplied in the form of yeast Marmite rice bran cereal bran wholemeal bread and milk As there may be faulty absorption 5 to 50 mg of crystalline vitamin $B_1$ may be injected intramuscularly or intravenously daily for twelve doses During the acute stages the patient must be kept in bed if there is venous engorgement relief is obtained by bleeding Massage should be given to the extremities

## Pellagra

**Definition** A disease characterised by dermatitis, gastro intestinal and nervous disturbances

**Etiology** Pellagra is believed to be due to deficiency of vitamin $B_2$ (nicotinic acid and riboflavin) vitamin $B_6$ (pyridoxin) and possibly vitamin $B_1$ The disease is associated with diets rich in maize but poor in meat and milk It is possible that maize contains anti vitamins

glycosuria probably results from reflex stimulation of the adrenals and liver through the splanchnic nerves

3 *Storage Deficiency* The liver may not be able to store completely the ingested glucose as glycogen with resultant hepatic glycosuria A lag curve (see Fig 63) indicates a defect in the storage mechanism or an abnormally rapid absorption In health it is very doubtful whether alimentary glycosuria can occur *i e* glycosuria after a meal rich in carbohydrate

4 *A Low Renal Threshold* If the renal threshold for glucose is below the normal of 180 mg glucose per 100 ml blood, renal glycosuria (see Fig 63) will ensue

In all cases in which glucose is found in the urine it is wise to determine the dextrose tolerance as this will indicate the severity of the condition

## Diabetes Mellitus

**Definition** A disease characterised by glycosuria hyperglycæmia and a disturbance of carbohydrate fat protein water and electrolyte metabolism

**Etiology** Diabetes mellitus is due to a deficiency of insulin the internal secretion of the pancreas *Predisposing causes* 1 Age Diabetes occurs at all ages but less commonly at the extremes of life 80% of all cases are over the age of 40 years 2 Sex Females predominate 3 Heredity There is a tendency to a familial incidence but the disease is not congenital 4 Race Jews are prone to diabetes 5 Habits Overeating especially of fats and lack of exercise Eighty per cent of diabetic patients are obese when the disease is first discovered but this does not apply to children 6 Nervous shock This is of doubtful significance

**Pathology** Insulin is derived from the islets of Langerhans In diabetes the blood sugar is above the normal range of 80 to 120 mg per 100 ml The sugar tolerance is diminished as shown by the curve of blood sugar readings obtained after giving 50 G of dextrose by mouth to the fasting patient and estimating the blood sugar every half hour Glucose is present in the urine and acetone and diacetic acid may also be found there *The pancreas* It is usually thought that diabetes mellitus results from damage to the $\beta$ cells of the islets of Langerhans The pathological changes however of fibrosis hyalinization and hydropic degeneration are only found to a marked degree in about 21% of cases and in about 23% the pancreas appears normal *The pituitary* The well known Houssay experiment showed that if a depancreatised dog is hypophysectomised the diabetes disappears and hypoglycæmia may ensue If an injection of an extract of the anterior lobe of the pituitary is now made into such a Houssay dog death will result from glycosuria and ketonuria This is the basis of the belief that diabetogenic and ketogenic hormones are formed in the anterior lobe and it is now believed that the growth hormone and the adreno corticotropic hormone are concerned with the production of glycosuria (see p 702) It was later shown that repeated injections of an extract

t i d or mg 100 taken by mouth t i d Nicotinamide (niacin amide) has the advantage over nicotinic acid of not causing flushing or abdominal pain In addition a mixed dietary should be given including fresh milk, meat liver eggs, fruit and no maize If there is a sore tongue and angular stomatitis riboflavin, mg 5 should be given t i d For diarrhœa and achlorhydria dilute hydrochloric acid should be given in m 30 to 60 doses diluted with 4 fl oz of water and a little syrup of orange three times a day after meals Injections of liver extract should also be given or stomach extract taken by mouth Peripheral neuritis should be treated by daily intramuscular injections of mg 50 to 100 of crystalline vitamin $B_1$ The skin should be protected from the sun, and for the moist lesions pot permang solution 1 in 5 000 should be applied Good results have also been obtained by intravenous injections of sod thiosulphate 10 ml of a 10% solution being given daily for 20 to 60 days

## Famine or War Œdema

A condition of œdema of the legs may be met with amongst prisoners of war due to a lack of protein in the diet or retention of sodium in the tissue spaces It is also occurs in civilian life when the chief article of diet is alcohol and in some cases of anorexia nervosa The plasma proteins are low and fluid passes by osmosis into the subcutaneous tissues

**Treatment** Blood transfusions of 300 to 500 ml should be given twice a week and an adequate intake of protein ensured

## Kwashiorkor

This disease which was described in the Gold Coast has a wide distribution in Africa Asia the West Indies South and Central America and it has been met with in Italy and Hungary It is common where malnutrition prevails amongst children It is due to lack of protein in the food and occurs usually between the ages of weaning and 4 years The hair loses its pigment becomes sparse and dry and may be reddish or white There is pallor local or general œdema enlargement of the liver apathy diarrhœa and as the disease progresses a reddish rash consisting of hyperkeratotic patches which desquamate The liver is fatty and the pancreas fibrotic The serum proteins are low Treatment consists in transfusions of plasma and the administration of skimmed milk the fat content being increased as soon as possible

## Glycosuria

Glucose is found in the urine as the result of alterations in the secretion of the ductless glands deficiency in the body storage mechanism or lowering of the renal threshold for sugar

**Etiology** 1 *Affections of the Ductless Glands* Pancreatic lesions causing deficiency in insulin as in diabetes mellitus or hæmochromatosis Hyperthyroidism Hyperpituitarism Hyperadrenia

2 *Cerebral Lesions* such as tumours hæmorrhage or meningitis and experimental piqure of the floor of the fourth ventricle The

reflexes tachycardia of over 120 a subnormal temperature low blood pressure a leucocytosis of 15 000 per c mm or over the urine contains sugar and ketone bodies and the alkali reserve of the blood is low the blood sugar is high When there is renal failure the urine may not give the ferric chloride reaction although the breath smells strongly of acetone

**Differential Diagnosis** The reducing substance in the urine may not be glucose Thus Fehling s solution is reduced by uric acid creatinine glycuronic acid lævulose lactose pentose and homogentisic acid

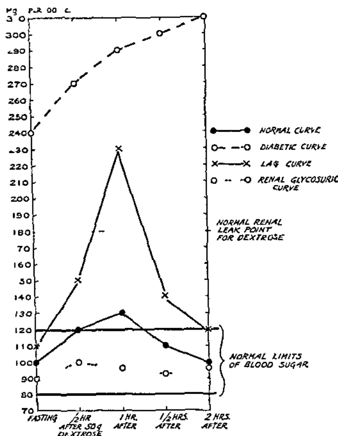



Fig 63 Chart showing Dextrose Tolerance Curves

Benedict s solution is not reduced by uric acid or creatinine Reduction with Benedict s solution therefore indicates a derangement of carbo hydrate metabolism with the exception of homogentisic acid and the actual substance present can be identified by special tests Glycosuria however does not necessarily mean diabetes A blood dextrose tolerance test will decide whether diabetes is present and if so its severity Typical results are shown in the graphs given (see Fig 63)

In renal glycosuria although there is glycosuria the blood sugar curve shows that the renal threshold is low, sugar appearing in the urine although the amount in the blood is not above normal Renal

of the anterior lobe will produce permanent diabetes in the dog which is not associated with ketonuria The pancreas in such a case shows an absence of islets *or a reduction* in the number of the $\beta$ cells *The liver* Usually no changes *are found* The metabolism of carbohydrate protein and fat is disturbed *The carbohydrate* which is absorbed from the intestines in the form of *monosaccharides* (glucose levulose and galactose) is not stored efficiently in the liver as glycogen and is not used adequately by *the muscles* but is excreted in the urine as glucose *Protein* The metabolism is disturbed as the sugar portion of glyco protein is excreted *and not* metabolised *Fat metabolism* Without efficient deposition of glycogen *in* the liver acetone bodies are formed in excess in the liver Acidosis or ketosis results, with diminution in the alkali *reserve in the* body and blood

**Clinical Findings** The patient is usually an adult who seeks medical advice for such symptoms as general weakness, wasting, pruritus vulvæ, balanitis thirst, polyuria, pains in the legs boils and carbuncles gangrene of a toe or constipation In some instances *failure of sight* or impotence is first complained of, or the patient may be seen for the first time in a state of coma

*On Examination* The patient may be well nourished (*diabete gras*) or thin (*diabete maigre*) The tongue may be red and dry and the complexion high coloured In the degenerative type of case the patient is usually over middle age and signs of cardio vascular degeneration are present The urine The specific gravity is usually over 1 020, and the reaction is acid A copper reducing substance is present as shown by Benedict s test Special laboratory tests demonstrate that the substance is glucose Acidosis may or may not be present, as shown by Gerhardt s ferric chloride test and the more delicate sodium nitro prusside test of Rothera The blood Sugar is present above the normal range of 80 to 120 mg per 100 ml in a specimen taken when the patient is not fasting the amount of blood sugar present is usually proportionate to the severity of the disease The alkali reserve indicates the presence or absence of acidosis The normal figure for the $CO_2$ capacity of the blood is 53 to 77 ml CO per 100 ml plasma With acidosis lower figures are obtained

*Coma due to Ketosis* Coma is very largely preventable It may be caused by the patient taking too much carbohydrate but more frequently it is due to an intercurrent infection which increases the amount of insulin required Another cause is an attack of gastro enteritis The patient then makes the mistake of reducing the insulin or taking none at all as he is having less food whereas really a larger dose of insulin is required Trauma or a surgical operation may also precipitate coma When a patient is ill the urine must be tested 3 or 4 times a day to make sure enough insulin is being given Diabetic coma does not come on suddenly there are always warning symptoms At the onset the patient may complain of abdominal pain constipation nausea vomiting or restlessness The chief clinical features during coma are air hunger (Kussmaul s type with slow and deep respirations) a smell of acetone in the breath flaccidity of muscles softness of the eyeballs loss of deep



Fig 64 Insulin Lipodystrophy

glycosuria may rarely develop later into diabetes a patient who has this condition should have a *sugar tolerance test* done once a year beyond this no treatment is required A " lag " curve may be obtained The sugar storage mechanism cannot keep pace with the dextrose absorption Glycosuria occurs after a meal rich in carbohydrates and the blood sugar curve shows a fall to normal in 2 hours A ' lag ' curve may also occur after gastro enterostomy and in some cases of duodenal ulcer owing to rapid absorption of dextrose from the intestine This condition may lead on to diabetes

Renal glycosuria must be distinguished from pentosuria In both conditions a substance is present in all specimens of the urine which reduces Benedict s solution and the blood sugar and sugar tolerance curves are normal Special chemical tests on the urine are required to distinguish between pentose and glucose the simplest being the *fermentation one Pentose does not ferment yeast whereas glucose does*

Coma due to ketosis must be differentiated from meningitis or cerebral hæmorrhage in which hyperglycæmia and glycosuria are often present If the temperature is normal or above normal the case is probably not one of uncomplicated diabetic coma

**Course and Complications** The course depends upon the type of disease present mild or severe, in children it is usually severe, in elderly people mild The course can be favourably modified by adequate treatment in the majority of cases Complications include Septic lesions such as boils carbuncles multiple abscesses in muscular tissues *and streptococcal cutaneous or subcutaneous lesions Insulin lipo* dystrophy occurring at the site of insulin injections (see Fig 64) especially in women and children This may cause great mental and physical distress The cause is unknown, as is also the cure Arterio sclerosis The Kimmelstiel Wilson kidney (see p 479) Pulmonary tuberculosis Gangrene of the lung Peripheral neuritis Myocardial degeneration with symptoms of heart failure Gangrene especially of the toes Coma either hyper or hypoglycæmic the former being due to acidosis the latter to overdosage of insulin Cataract Retinopathy Korsakow s psychosis (see p 778)

*Hypoglycæmia The patient who is receiving insulin for the* treatment of diabetes may suffer from hypoglycæmia With soluble insulin this is particularly liable to occur if a carbohydrate meal is not taken within half an hour of the injection or if the patient takes an undue amount of exercise The early symptoms are sweating flushing or pallor hunger headache abdominal discomfort weakness tremors visual disturbances and coldness of the extremities Consciousness may be retained although the patient is unable to move or speak The patient may vomit and become unconscious with marked convulsions resembling those of acute mania Hemiplegia sometimes occurs *Children may* become pale and then suddenly pass into coma The hypoglycæmic attack may not occur until about 4 p m *i e*, after lunch although no insulin has been given since before breakfast The treatment is described later (see p 668)

**Prognosis** This is good in all mild types of disease *in severe*

cases the prognosis is usually favourable provided the patient can be adequately treated and will be conscientious in his after treatment The diabetic death rate in Great Britain fell by about 25% to 30% in all age groups between 1939 and 1945 This may have been due to better treatment rationing or a lower incidence of the disease Given skilled stabilising treatment the prognosis largely depends upon the patient Perhaps in no other disease does a good patient reap such a rich reward Any septic focus may predispose to or excite coma Diabetes with tuberculosis in the past formed a serious combination and the patient usually required a large amount of insulin With the use of streptomycin isoniazid and para aminosalicylic acid in the treatment of tuberculosis the diabetes is much more easily controlled The insulin required often falls in patients who are meticulous in carrying out the dieting, and in some cases after 2 or 3 years no insulin is required Death in young people is usually due to coma Other causes of death are cardio vascular degeneration sepsis and infection without coma and intercurrent diseases especially tuberculosis and cancer

Treatment *Stabilising Treatment* Except in very acute cases in which it is essential to begin insulin treatment immediately a dextrose tolerance test should be made after giving the patient a diet containing 300 G carbohydrate for 3 days This will confirm the diagnosis of diabetes and give some idea of its severity Mild cases in adults and diabetes in middle aged obese patients may often be treated by dieting without insulin In children in severe cases in adults and in any case in which complications are present such as ketosis peripheral neuritis diabetic retinopathy gangrene of the extremities and pulmonary tuberculosis insulin should be administered at once

*Starvation followed by graduated diets* This is used in the minority of cases particularly when the disease is mild or if the patient refuses insulin No patient with ketosis should be starved as there is a grave risk of precipitating coma A patient who is being starved is put to bed and given fluids such as water lemonade sweetened with saccharin Bovril and weak tea or coffee without sugar or milk The urine usually becomes sugar free in about 36 hours The patient is then worked up through a series of graduated diets until he is taking an adequate diet If the urine remains sugar free well and good if not insulin will be required The diets are increased by about 10 G of carbohydrate a day The adequate diet is calculated by determining the number of calories required on the basis of 15 cals for 1 lb correct body weight and adding 10 to 20% more to the figure for the basal requirement diet so obtained according to the muscular work which will be done when the patient is living a normal life The correct body weight may be obtained from age sex and height tables

*Insulin Treatment* The patient is given a diet the calorie value of which depends upon the estimated requirements for his normal activites This usually contains about 200 G carbohydrate 90 to 100 G protein and 90 G fat The calorie value can be easily worked out as 1 G of carbohydrate equals approximately 4 cals 1 G protein 4 cals and 1 G fat 9 cals An example of such a diet is as follows —

| | | G | | | mg./100 G | | Cals |
|---|---|---|---|---|---|---|---|
| | | C | P | F | Na | K | |
| *Fresh Meat* | | | | | | | |
| Rabbit (stewed) | 1 oz. | 0 | 8 | 2.3 | 47 | 370 | 53 |
| Hare (stewed or roast) | 1 | 0 | 8.8 | 2.4 | 40 | 211 | 57 |
| Veal (cutlet fried) | 1 | 0 | 9.1 | 2.4 | 48 | 330 | 58 |
| Beef (roast) | 1 | 0 | 8 | 3.7 | 70 | 357 | 65 |
| Veal (fillet roast) | 1 | 0 | 9.2 | 3.5 | 53 | 380 | 68 |
| Mutton (boiled) | 1 | 0 | 7.7 | 5 | 110 | 330 | 76 |
| Mutton (roast) | 1 | 0 | 7.5 | 6.1 | 71 | 340 | 85 |
| Pork (leg roast) | 1 | 0 | 7.4 | 7 | 58 | 200 | 93 |
| Pork (loin roast) | 1 | 0 | 7.8 | 10.7 | 69 | 353 | 128 |
| *Poultry and Game* | | | | | | | |
| Grouse (roast) | 1 | 0 | 9 | 1.6 | 96 | 466 | 50 |
| Chicken (roast) | 1 | 0 | 8.9 | 2.2 | 110 | 250 | 55 |
| Turkey (roast) | 1 | 0 | 9 | 2.3 | 92 | 310 | 57 |
| Chicken (boiled) | 1 | 0 | 7.9 | 3.1 | 98 | 381 | 60 |
| Guinea fowl (roast) | 1 | 0 | 9.8 | 2.5 | 136 | 430 | 62 |
| Partridge (roast) | 1 | 0 | 10.6 | 2.2 | 100 | 407 | 62 |
| Pheasant (roast) | 1 | 0 | 9.2 | 2.8 | 104 | 411 | 62 |
| Pigeon (roast) | 1 | 0 | 8 | 4 | 105 | 410 | 68 |
| Duck (roast) | 1 | 0 | 6.8 | 7.1 | 96 | 210 | 91 |
| Goose (roast) | 1 | 0 | 8.4 | 6.7 | 145 | 406 | 94 |
| *Internal Organs* | | | | | | | |
| Brain (calf sheep boiled) | 1 | 0 | 3.5 | 1.9 | 150 | 340 | 31 |
| Kidney (stewed) | 1 | 0 | 7.7 | 1.7 | 210 | 310 | 46 |
| Sweetbread (stewed) | 1 | 0 | 6.8 | 2.7 | 69 | 231 | 52 |
| Kidney (fried) | 1 | 0 | 8.4 | 2.7 | 261 | 304 | 58 |
| Liver (calf fried after rolling in flour) | 1 | 0.7 | 8.7 | 4.4 | 110 | 380 | 77 |
| Tongue (sheep fresh stewed) | 1 | 0 | 5.5 | 7.2 | 100 | 260 | 87 |
| *Fish* | | | | | | | |
| Oysters | 1 | 0 | 3 | 0.3 | 73 | 110 | 15 |
| Cod (steamed) | 1 | 0 | 5.4 | 0.3 | 100 | 360 | 24 |
| Sole (steamed) | 1 | 0 | 5.3 | 0.4 | 110 | 240 | 25 |
| Lemon sole (steamed) | 1 | 0 | 6 | 0.3 | 115 | 279 | 27 |
| Plaice (steamed) | 1 | 0 | 5.4 | 0.6 | 120 | 278 | 27 |
| Whiting (steamed) | 1 | 0 | 6 | 0.3 | 2[illegible]5 | 112 | 27 |
| Haddock (steamed) | 1 | 0 | 6.6 | 0.2 | 121 | 323 | 28 |
| Turbot (steamed) | 1 | 0 | 6.2 | 0.5 | 90 | 285 | 29 |
| Haddock (smoked steamed) | 1 | 0 | 6.7 | 0.3 | 1 220 | 293 | 30 |
| Hake (steamed) | 1 | 0 | 5.5 | 1 | 118 | 310 | 31 |
| Lobster (boiled) | 1 | 0 | 6.4 | 1 | 325 | 258 | 35 |
| Trout (river steamed) | 1 | 0 | 6.7 | 1 | 88 | 374 | 36 |
| Crab (boiled) | 1 | 0 | 5.8 | 1.6 | 366 | 271 | 38 |
| Halibut (steamed) | 1 | 0 | 6.8 | 1.2 | 56 | 540 | 38 |
| Mullet (red steamed) | 1 | 0 | 6.5 | 1.3 | 118 | 364 | 38 |
| Mullet (grey steamed) | 1 | 0 | 6.5 | 1.3 | 94 | 275 | 38 |
| Salmon (tinned) | 1 | 0 | 5.9 | 1.8 | 538 | 320 | 40 |
| Herring (baked in vinegar) | 1 | 0 | 5 | 3.9 | 62 | 233 | 55 |
| Salmon (steamed) | 1 | 0 | 5.7 | 3.9 | 48 | 410 | 58 |
| Kipper (baked) | 1 | 0 | 7 | 3.4 | 990 | 520 | 59 |
| Sardines (tinned) | 1 | 0 | 6.1 | 6.8 | 760 | 260 | 86 |
| *Miscellaneous* | | | | | | | |
| Pickles (mixed unsweetened) | 1 | 1.2 | 0.2 | 0.1 | 2 300 | 300 | 25 |
| Olive oil | 1 | 0 | 0 | 30 | 0.2 | 0.2 | 270 |
| Whisky ([illegible]5 % alcohol) | 1 | 0 | 0 | 0 | 0.1 | 0.6 | 50 |
| Lump sugar (8 lumps ⅝ inch cube) | 1 | 30 | 0 | 0 | 0.3 | 0.5 | 120 |

**Breakfast** Tea or coffee Milk, 4 oz , Bread, 2 oz Oatmeal (dry) 1 oz Butter ¼ oz Marmalade ¼ oz Egg 1 **C** 63 5 **G** **P** 21 **G** **F** 27 **G** **Cals** 581 **11 a m** Tea or coffee Milk 1 oz Plain biscuit 1 **C** 6 5 **G** **P** 1 **G** **F** 1 **G** **Cals** 38 **Lunch** Fish 3 oz Potatoes 2 oz Bread 1 oz Butter ¼ oz , Milk 5 oz Stewed apples 4 oz Cheese (Cheddar) 1 oz **C** 3[illegible] **G** **P** 33 **G** **F** 27 **G** **Cals** 515 **Tea** Tea Milk 2 oz , Bread 1¼ oz Butter ¼ oz Salad if desired **C** 24 **G** **P** 5 **G** **F** 8 **G** **Cals** 188 **Dinner** Clear soup if desired Lean meat 3 oz Potatoes (boiled) 2 oz Butter ¼ oz Green vegetables 2 oz Bread 2 oz Milk 7 oz Cheese (Cheddar) 1 oz **C** 56 **G** **P** 45 **G** , **F** 33 **G** **Cals** 701 **Bedtime** Milk 4 oz Plain biscuits 2 **C** 16 **G** **P** 4 **G** **F** 4 **G** **Cals** 116 **Total** **C** 201 **G** **P** 109 **G** **F** 100 **G** **Cals** 2 139

In hospitals to which dietitians are attached the diet will be supplied according to the patient's requirements the actual distribution of carbohydrate between the four meals being varied according to whether soluble insulin is being given in 1 or 2 doses morning and evening or whether a combination of soluble insulin with protamine zinc insulin or a lente insulin is injected in one morning dose The

**Food Analyses**

| | | G | | | mg /100 G | | Cals |
|---|---|---|---|---|---|---|---|
| | | C | P | F | Na | K | |
| *Farinaceous Foods* | | | | | | | |
| Bread (white) | 1 oz | 14 3 | 2 | 0 4 | 393 | 70 | 69 |
| Bread (brown) | 1 | 13 | 1 4 | 0 4 | 393 | 147 | 61 |
| Toast (white) | 1 | 17 2 | 2 3 | 0 3 | 467 | 83 | 81 |
| Toast (fairy) | 1 | 21 | 3 | 0 6 | — | — | 103 |
| Energen bread (1 roll) | — | 2 | 1 [illegible] | 0 2[illegible] | — | — | 16 |
| Ryvita (3 sections) | 1 | 21 2 | 3 3 | 0 4 | 615 | 469 | 10[illegible] |
| Vitaweat (3 sections) | 1 | 21 2 | 3 3 | 2 2 | 60[illegible] | 430 | 121 |
| Rice (uncooked) | 1 | 22 7 | 1 6 | 0 1 | 6 3 | 113 | 98 |
| Oatmeal (dry) | 1 | 18 | 4 0 | 2 | 33 4 | 368 | 108 |
| *Dairy Produce* | | | | | | | |
| Egg (1) | 2 | 0 | 7 6 | 4 8 | 140 | 130 | 74 |
| Milk | 1 | 1 5 | 1 | 1 | 51 | 140 | 19 |
| Butter | 1 | 0 | 0 | 2[illegible] | [illegible]80 | 60 | 22[illegible] |
| *Cream (20%)* | 1 | 1 | 1 | 6 | 40 | 56 | 62 |
| Cheese | | | | | | | |
| Camembert | 1 | 0 | 6 3 | 6 5 | — | — | 84 |
| Dutch | 1 | 0 | 9 2 | [illegible] 3 | 1 2[illegible]0 | 96 | 84 |
| Wensleydale | 1 | 0 | 10 | 6 | — | — | [illegible]4 |
| Gorgonzola | 1 | 0 5 | 7 8 | 8 | 1 220 | 172 | 10[illegible] |
| Gruyere | 1 | 0 | 9 4 | 8 [illegible] | 542 | 125 | 114 |
| Cheddar | 1 | 0 9 | 10 | 8 | 540 | 130 | 116 |
| Cheshire | 1 | 1 3 | 8 8 | 9 2 | — | — | 1[illegible]3 |
| St Ivel | 1 | 0 | 7 1 | 10 [illegible] | 567 | 68 | 123 |
| Roquefort | 1 | 0 [illegible] | 10 4 | 9 4 | — | — | 1 8 |
| Stilton | 1 | 0 | 7 2 | 11 7 | 1 1[illegible]0 | 161 | 134 |
| *Preserved Meals* | | | | | | | |
| Bacon (back fried) | 1 | 0 | 7 4 | 16 | 760 | 9[illegible] | 174 |
| Bacon (streaky fried) | 1 | 0 | 7 2 | 13 8 | 3 000 | 162 | 153 |
| Ham (lean boiled) | 1 | 0 | 6 9 | 4 | 2 100 | [illegible]10 | 64 |
| Tongue (ox canned) | 1 | 0 | 5 8 | 7 | 100 | 260 | 86 |

in two strengths 40 and 80 units per ml If zinc insulin is given the carbohydrate content of the food should be divided approximately equally between the four meals and about 10 G of carbohydrate taken at bedtime

If protamine zinc insulin is injected before breakfast it is often found that the noon specimen of urine contains sugar whereas specimens later in the day and in the early morning are sugar free In such cases soluble insulin should be injected before breakfast with the protamine zinc insulin and the dose of soluble insulin should always be slightly greater than that of the zinc insulin The two insulins are put in the same syringe and thus only one injection is required Care must be taken to avoid mixing as far as possible the soluble and protamine zinc insulins in the syringe as the soluble insulin is thereby inactivated by the zinc insulin An amount of air equal to the volume of zinc insulin required is first injected into the zinc insulin phial The needle is withdrawn and the required amount of soluble insulin is drawn into the syringe The zinc insulin is now drawn into the syringe taking care that no air enters the syringe The injection is now given Owing to the slow rate of absorption of zinc insulin it is useless for the treatment of coma associated with hyperglycæmia for which soluble insulin alone is efficient

*Insulin zinc suspensions* do not contain protein Many adults are now treated with one of the insulin zinc suspensions The insulin action is delayed by the addition of zinc and an acetate buffer The duration of the action of the insulin depends on the size of the suspended particles thus amorphous insulin has a shorter action than crystalline insulin There are three varieties of insulin zinc suspension I Z S (amorphous) or semilente insulin is effective for about 10 to 15 hours I Z S (crystalline) or ultralente insulin has a delayed action which persists for about 24 to 36 hours I Z S or lente insulin consists of a mixture of three parts of amorphous and seven parts of crystalline insulin This is the favourite variety and it produces a fairly continuous lowering of the blood sugar for 24 hours after injection The carbohydrate content of the food should be arranged so that the evening meal contains less than breakfast or the midday meal If more than 50 units are required it is better to use a combination of soluble insulin with protamine zinc insulin The lente insulins are injected in the morning on getting up Insulin zinc suspensions are put up in 40 and 80 units per ml in 10 ml vials The vial must be well shaken and the injection quickly made They seldom provoke local insulin reactions being free from protein They are not effective in severe cases of hyperglycæmia with ketosis especially when there is intercurrent infection

*The Use of Oral Sulphonamides* Certain sulphonamide preparations have been discovered to have a hypoglycæmic effect when given by mouth A substance called BZ 55 is on trial It is N butyl N sulphanilylurea also called Bucrol Nadisan or Carbutamide It is put up in 0 5 G tablets The average dose for an adult is 2 5 G the first day 1 5 G the second day and then 0 5 G morning and evening It has been found effective in controlling diabetes in the middle aged or

medical student and doctor should be familiar with the constitution and calorie value of the various food stuffs so that he can draw up the diet himself, if necessary In acute cases, and in children, better results are obtained by the use of high carbohydrate diets, containing 250 to 300 G carbohydrate The fat should be limited to about 90 G and the remainder of energy required is supplied by protein An example of a high carbohydrate diet is as follows —

7 a m 2 biscuits ($\frac{1}{2}$ oz ) **Breakfast** Apple 2 oz Egg, 1 , Bread 1 oz Oatmeal (dry), 1 oz , Jam $\frac{1}{4}$ oz , Milk 6 oz Sugar $2\frac{1}{2}$ level teaspoonfuls Mid morning Bread 1 oz or 3 biscuits ($\frac{3}{4}$ oz ) Lunch Bovril 10 oz Lean meat, $1\frac{1}{2}$ oz , Potato (cooked), 3 oz , Butter, $\frac{1}{3}$ oz , Apple, 2 oz Vegetable Group A 3 oz Bread, 1 oz Tea Milk 4 oz Bread 2 oz Plain cake, 1 piece (2 oz ), Jam $\frac{1}{2}$ oz Banana, 1 or bread 1 oz **Supper** Fish $1\frac{1}{2}$ oz Potato (cooked) 3 oz , Butter, $\frac{1}{3}$ oz Apple 2 oz , Milk 4 oz Vegetable Group A , 3 oz Bread, 1 oz , Bananas 2 or Bread 2 oz **10 p m** Milk 4 oz Plain cake, 1 piece (2 oz ) or 3 biscuits ($\frac{3}{4}$ oz ) C 309 G P 81 G , F 68 G Calories = 2 172

*The Administration of Insulin* It is not usually necessary for blood sugar estimations to be made daily during the preliminary stabilising treatment providing the urine is tested regularly for sugar The following routine may be employed 8 a m urine collected and insulin injected if sugar is present 8 30 a m breakfast 12 noon urine collected 1 p m lunch 3 30 p m urine collected 4 30 p m tea 7 p m urine collected and insulin injected if required 7 30 p m dinner 9 p m urine collected The specimen passed before insulin is due must always be tested before insulin is injected The insulin is gradually increased until the necessary amount is determined Five units of soluble insulin are injected half an hour before breakfast and the insulin is increased 5 units at a time every 2 or 3 days until the midday specimen of urine is sugar free If now the 8 a m specimen of urine contains sugar an evening dose of insulin is given half an hour before dinner and the dose increased by 5 units every 2 or 3 days until the before breakfast specimen is sugar free

*Soluble insulin* is put up in strengths of 20 40 and 80 units per ml It has a rapid action beginning about half an hour after injection reaching its maximum in about 3 hours and ceasing to have any effect in 6 hours With soluble insulin breakfast contains the greatest amount of carbohydrate dinner the next largest amount, with relatively little at lunch and tea Not more than 50 units of soluble insulin should be given at a single injection except in the treatment of coma Soluble insulin is necessary for the treatment of hyperglycæmic coma before or after surgical operations in acute gastro enteritis and for children or young people in whom rapid changes occur in the level of the blood sugar

*Protamine zinc insulin* has a delayed effect beginning about 6 hours after injection and lasting up to 24 hours or longer It therefore tends to have a cumulative effect if daily injections are given, and is more likely to lead to hypoglycæmic coma than is soluble insulin It is put up

containing meat within half an hour of a dose of insulin that he should not take violent exercise while he is having injections that he should carry sugar with him to eat should early symptoms of hypoglycæmia occur If he gets a feverish cold his doctor should be informed as he will temporarily require more insulin He must always guard against constipation He may also be given a table showing how his standard diet can be varied

## Treatment of Coma

**Coma Due to Ketosis** The following routine should be observed The patient should be admitted to hospital if possible The bed should be warmed with an electric blanket or hot bottles great care being taken to avoid burning the patient The lungs should be examined to exclude pneumonia and if present a course of penicillin injections begun A catheter specimen of urine is now taken and tested for glucose and ketone bodies A specimen of blood is taken from an antecubital vein and sent to the laboratory for blood sugar urea and alkali reserve estimations The blood pressure is taken If the urine contains much sugar give an immediate injection of 100 units of insulin subcutaneously and if the pulse is very feeble 20 units also intravenously There is usually considerable dehydration owing to the polyuria and possibly to the vomiting in the early stages Five litres or more of fluid may be lost from the blood and tissue spaces the greater part being from the latter site To combat dehydration give an intravenous injection of 1 500 ml of normal saline in 1 hour and later run it in at the rate of 500 ml an hour up to a total of 4 to 5 litres in 24 hours If there is evidence of cardiac failure the saline must be run in more slowly taking care that œdema of the lungs does not develop Alternatively a solution of sod chlorid 5 85 G sod lact 3 36 G aq dest ad 1 000 ml may be used The stomach should be washed out and an enema given if the patient is not too collapsed The blood sugar reading should now be available but if this is not possible subsequent insulin injections must be guided by the sugar content of the urine Some authorities advise injecting 100 to 200 units of insulin every hour at first but usually good results are obtained with 50 units every hour The mistake which is liable to be made is not to give enough insulin In very severe cases larger amounts are required up to 1 000 units in the first 24 hours As soon as ketone bodies disappear from the urine smaller amounts of insulin are required It is a moot point whether or not dextrose should be injected intravenously By so doing ketosis is likely to disappear more rapidly and it also avoids the possibility of converting the hyperglycæmia into hypoglycæmia It is usually helpful to replace the normal saline drip by one containing 5% dextrose in one third normal saline after the first 2 to 3 litres have been infused running it in at 500 ml an hour This also usually increases the urine output and aids in raising the blood pressure If the pressure remains low 500 ml of whole blood should be given intravenously In addition Coramine 1 5 to 5 ml may be injected into a vein If the urine shows no glucose at any test no further insulin should be given until a blood sugar reading has been obtained In some

elderly diabetic who has never had ketosis and who needs less than 30 units of insulin a day It should only be used under careful hospital supervision as there is a danger of severe agranulocytosis and of thrombocytopenia Tolbutamide is also available in similar doses and is apparently much less toxic

When the patient is on a satisfactory diet a careful search should be made for septic foci in the mouth, nose and throat, etc and if found they should be eradicated a chronically inflamed appendix or gall bladder may require removal before the metabolic processes are properly stabilised

*Diabetic Treatment in case of Operations* If an operation is required it should be performed under a local anæsthetic or with the aid of gas and oxygen thiopentone cyclopropane etc Chloroform and ether should never be given The patient should be stabilised as described above and not starved before the anæsthetic He should have his last meal 3 to 4 hours before the operation Two and a half hours before the anæsthetic he should be given 30 units of insulin and half an hour later dextrose oz 2 dissolved in water fl oz 6 by mouth On recovery from the anæsthetic the patient is given dextrose oz 5 to 6 by mouth or intravenously during the first 24 hours Thus dextrose oz 2 dissolved in water fl oz 6 may be given 3 times during the 24 hours with 10 to 20 units of insulin injected half an hour before each dose No insulin should be injected unless the blood sugar is above 120 mg per 100 ml or if the urine is sugar free Four pints of fluid in all must be given during the first 24 hours either by mouth or intravenously The next day the patient will probably be able to take citrated milk feeds of 4 to 6 fl oz every 3 hours with dextrose oz 1 in three of the feeds Half an hour before these dextrose feeds 10 to 20 units of insulin should be injected The urine must be tested for sugar before each insulin injection The patient is then worked up again through the diets but the earlier ones can usually be omitted

*Diabetes and Pregnancy* A diabetic woman can safely be allowed to become pregnant if she is properly treated Further pregnancy is more likely to occur in a diabetic who is under treatment with insulin than in one who is not being so treated Still birth is common in diabetes unless the disease is accurately controlled The blood sugar of an infant born of a diabetic mother is often low (4 to 22 mg per 100 ml ) and hypoglycæmic convulsions may occur which are quickly relieved by the administration of dextrose During pregnancy there is a tendency to ketonuria When the patient is in labour milk dextrose and insulin should be given as described under the treatment in the case of operations Cæsarean section is unnecessary During the puerperium there is a tendency to hypoglycæmia and the insulin dosage may be reduced

*After Treatment* When the diet and the necessary insulin dosage are determined the patient can begin to look after himself with monthly tests by the doctor of the blood and urine He should be instructed how to give the insulin how to sterilise the syringe and needles and how to test the urine morning and evening he should also be warned of the danger of hypoglycæmia that he must always have a carbohydrate

certain degree with the predisposing causes they include headache faintness nausea vomiting abdominal pain air hunger neuritis and pyrexia

**Differential Diagnosis** This is effected by 1 Examination of the urine for acetone bodies 2 Determination of the alkali reserve of the blood 3 Determination of the alveolar $CO_2$ tension

**Treatment** *Prophylactic* Acidosis can be prevented by ensuring a sufficiency of carbohydrate in the diet together with insulin if required in diabetes to metabolise the carbohydrate

*Curative* Dextrose must be given by mouth oz 1 to 2 daily or by rectum or intravenously so that the glycogen content of the liver is maintained Dehydration and salt depletion should be corrected by the intravenous injection of normal saline or of a mixture of isotonic sodium chloride and of isotonic sodium lactate

## Alkalosis

Alkalosis is characterised by an increase in the available alkali reserves in the blood In alkalæmia the hydrogen ion concentration is lowered

**Clinical Findings** Alkalosis results from administration of massive doses of alkali as in the treatment of gastric or duodenal ulcers especially if there is gastric stasis or renal insufficiency It may also be caused by persistent vomiting due to obstruction at the pylorus or high up in the intestine In hyperventilation and in anoxæmia associated with high altitudes or heart failure there is alkalæmia with a fall in the alkali reserves The chief symptoms are Malaise headache giddiness anorexia vomiting tetany and coma

**Differential Diagnosis** This can be confirmed by determining the alkali reserve of the blood and the blood nitrogen figures are usually raised

**Treatment** All alkalis should be discontinued at once and 1 oz of dextrose given by mouth 3 times a day Normal saline should be injected intravenously In severe cases the electrolytic balance must be restored by the intravenous injection of a solution of potassium sodium and ammonium chloride

## Gout

**Definition** A disease characterised by excess of uric acid in the blood uratic deposits in tissues and joint manifestations Podagra implies gout in the foot and cheiragra gout in the hand

**Etiology** The cause is not known There is possibly a metabolic error with regard to nucleoproteins combined with a sensitiveness to certain protein substances such as are contained in foods or bacteria which precipitate an attack Gout is often associated with asthma, urticaria or eczema diseases due in some instances to a toxic idiopathy Gout is not due solely to excess of uric acid in the blood as much higher figures are obtained in such diseases as leukæmia without any manifestation of gout It may be due primarily to a pituitary adrenal disturbance in view of the response to ACTH and cortisone *Predisposing causes*

cases after the ketosis has disappeared the blood potassium falls to a low level with extreme weakness of the limb muscles and of the diaphragm resulting in dyspnœa For this 2 G of potassium chloride may be given every hour for 3 doses if the patient can swallow or 200 ml of a 1% solution of potassium chloride are run slowly into a vein Potassium salts should not be given if there is oliguria

On recovery from coma the patient should be given milk fl oz 6 to 8, every 3 hours with water, meat extracts or orange juice between the milk feeds, and insulin 3 times a day in amounts sufficient to keep the urine nearly free from sugar acetone bodies will reappear and to get rid of them dextrose should be given in addition oz ½ to 1 in the three feeds which follow the insulin injections In about 24 to 48 hours the patient can be put on a diabetic diet and the extra dextrose continued until the acetone bodies disappear from the urine the insulin being adjusted to keep the urine sugar free A careful search for a septic focus should now be made and if possible it should be eradicated

**Coma Due to Hypoglycæmia** Hypoglycæmic coma may come on suddenly the patient having convulsions It may occur during the night while the patient is asleep The patient who is receiving insulin should always have with him some lumps of sugar and eat them at the earliest symptom Hypoglycæmic coma can usually be relieved by the intramuscular injection of 1 ml of liq adrenalin hydrochlor and the intravenous injection of 20 to 100 ml of 10% dextrose in normal saline which should be repeated every half hour if necessary In some cases as much as 200 G of dextrose are required Alternatively, if the coma is not very deep dextrose oz 2 in water fl oz 6 may be introduced into the stomach through a nasal tube When the patient can swallow he should be given dextrose oz ½ by mouth every hour for several hours especially if the hypoglycæmia is due to a protamine insulin

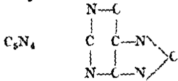
## Acidosis

Acidosis implies that there is a lowering of the alkali reserve (chiefly bicarbonates) in the blood There is not necessarily an acidæmia *i e*, increase in the hydrogen ion concentration of the blood In ketosis such abnormal substances as β hydroxybutyric acid and acetone are present in the blood

**Clinical Findings** Acidosis is of importance in several conditions such as Diabetes this is described on p 657 Unexplained pyrexia in children and recurrent cyclic vomiting may be due to acidosis such children are abnormally intolerant of fat in their diet or of fat containing medicines such as cod liver oil Von Gierke's disease is accompanied by acidosis Acidosis may occur in renal failure owing to diminished formation of hydrogen and ammonia ions and reduced urine output Severe starvation or chronic diarrhœa lead to acidosis Carbon monoxide poisoning causes acidosis by interference with the oxygen supply of the tissues In acute yellow atrophy of the liver and in pernicious vomiting of pregnancy acidosis may occur, probably due to the toxic effects on the liver Acidosis also results from the administration of ammonium chloride or of Diamox The symptoms vary to a

1 Heredity The gouty diathesis is frequently noted in Europe and it is said that in America out of 7 million sufferers from chronic rheumatism one third of a million are affected by gout 2 Age Usually middle age and after 3 Sex Males predominate 4 Food and alcohol Overeating especially of meat and overdrinking especially of beer port and sweet wines 5 Occupation Workers in lead and maltsters are prone to gout 6 Infection In teeth tonsils nasopharynx and the intestinal tract 7 Season Especially in the spring 8 Locality Chiefly in temperate zones especially England and Germany gout is rare in Scotland probably due to a low incidence of beer and wine drinking An attack may be precipitated by trauma to a joint by a surgical operation by injection of crude liver preparations by mental worries by certain articles of diet which vary in different individuals such as strawberries white wine port etc and by infection with micro organisms Gout is much less common nowadays presumably due to moderation in food and drink and the greater attention given to the eradication of septic foci

**Pathology** There is an excess of uric acid in the blood the average amount in gout being 5 to 9 mg per 100 ml (normal 1 to 4 mg per 100 ml plasma) Deposits of mono sodium urate are found in tissues which are comparatively avascular and rich in sodium Thus tophi ( chalk stones composed mainly of urates) occur in the cartilage of the ear eyelid nose in the olecranon and patella bursæ around joints especially of the hands and feet and in tendons and ligaments The uric acid elimination in the urine is diminished for a day or so before an acute attack of gout but increases during the attack Uric acid is derived from nucleoprotein nucleoprotein is present in the cell nuclei of the tissues (endogenous source) and in the nuclei of foodstuffs (exogenous source) It may also be synthesised to a slight degree in the body from glycine serine ammonia and carbon dioxide Nucleoproteins in the body are metabolised with the formation of nucleic acid a substance




containing the purin ring This is converted into adenine (amino purin) and guanine (diamino purin) and by oxidation into hypoxanthine (oxypurin) xanthine (dioxypurin) and finally into uric acid (trioxypurin) It is now considered probable that all the uric acid formed in man is not excreted some being further katabolised in the blood

Post mortem examination of an affected joint shows whitish smears of urate on the cartilage just under the surface the palms of the hands may show white lines the joints are deformed tophi may be seen ulcerating through the skin Death usually results from complications due to cardio vascular degeneration or chronic nephritis Thus myocardial degeneration pericarditis atheroma arteriosclerosis cerebral hæmorrhage chronic nephritis or bronchitis may be found Uratic deposits may be seen at the apex of the pyramids of the kidneys

**Clinical Findings** *Acute Gout* The patient is usually a middle aged

man, who may give a history of previous attacks or of digestive troubles For a few days before the attack he feels irritable depressed and may have flatulent dyspepsia and constipation The attack usually begins during the night the patient waking with severe pain of a burning boring character in the metatarso phalangeal joint of the big toe in the ankle or heel he may shiver and sweat and after several hours agony fall asleep to awake in the morning and find the joint swollen red hot tense and shiny Post operative gout is described appearing from a few hours to a few days after a surgical operation

*On Examination* During the attack the temperature is usually raised to about 100 to 102 F There is œdema around the affected part the neighbouring lymphatic glands may be enlarged and the veins engorged The pain usually recurs every night for 2 or 3 nights and the attack then passes off The skin may desquamate over the joint which feels numb At times the attack begins during the day or it may begin at night and last through the day After the attack the patient often feels extremely fit Other joints less frequently affected are those of the finger or thumb the knee wrist or elbow but very rarely the hip or shoulder Polyarthritic attacks are infrequent The blood During an attack this shows a polymorphonuclear leucocytosis of 20 000 to 25 000 per c mm The sedimentation rate of the red cells is considerably increased during the acute stage The urine Uric acid excretion falls before and rises during the attack for 2 or 3 days

**Differential Diagnosis** Acute gout must be distinguished from acute rheumatism suppuration around or in a joint gonorrheal arthritis infective arthritis synovitis an inflamed bunion and tuberculous or syphilitic arthritis The characteristics of acute gout are the typical appearances and history the presence of tophi in the ears (see Fig 65) etc and a blood uric acid content of over 4 mg per 100 ml

**Course and Complications** A second attack may never occur but usually it develops within a year subsequent recurrences are more frequent Complications include suppuration around tophi cardio vascular degeneration and chronic nephritis are often met with

**Prognosis** Gout is seldom fatal unless associated diseases are present Death is usually due to cardio vascular or renal lesions

**Treatment** *Prophylactic* When there is gout in the family the precautions required are the eradication of all septic foci and moderation in eating and drinking If the patient recognises the premonitory symptoms of an attack he should take tab colchicine gr 1/240 2 tabs two hourly for 4 to 6 doses

*Curative* During the attack The leg or arm should be supported on a pillow covered with wool and guarded by a cradle Warm *fomentations of lotio plumbi c opio (B P C)* on lint or of Sod bicarb oz 1 tnc opii fl oz 1 and water to a pint may be applied The bowels should be opened by a saline aperient At the first symptom colchicine should be given in the doses recommended above It should be con*tinued until the pain is relieved* or until diarrhœa occurs The diarrhœa should then be checked by tnc opii camphorat m 60 in water taken after each loose bowel action In a subsequent acute attack the



Fig 6 Ear showing Gouty Tophi



Fig 66 Ulcerating Tophi in Gout

4 mg/100 ml After the patient has been taking the tablets for a few weeks he may develop an attack of gout, the so called "Benemid gout" This may be prevented by the simultaneous administration of tab colchicine gr 1/240, 2 tabs daily for a few weeks at the onset of treatment

The alternative treatment is the administration of sodium salicylate gr 90 in divided doses daily for 6 months, when the dose can be reduced to three times a week

**Irregular Gout** (Suppressed or retrocedent gout) In some cases an acute attack of gout ceases suddenly as may happen if the affected part is put into cold water The patient may become unconscious or die Death is probably not due to gout but to associated degeneration in other organs such as the cardio vascular or renal systems It is thought that in some cases gout may give rise to acute gastric or intestinal symptoms to præcordial pain unrelated to effort and that ocular lesions such as iritis, conjunctivitis and choroiditis, may, in some cases be due to gout rather than to infection

## Obesity

## (*Lipomatosis Universalis*)

**Definition** Excessive generalised deposition of fat in the body

**Physiology and Pathology** The nature of the regulating mechanism which controls body weight is not known The diet of an average healthy male not doing heavy muscular work is composed of about 500 G of carbohydrate 100 G of protein and 100 G of fat and yields 3 300 calories In order to maintain a steady weight the energy output must balance this intake The energy expended is made up of basal metabolism metabolism due to muscular activity and possibly metabolism resulting from the specific dynamic action of food As a diet of 3 300 calories yields more energy than is necessary for physiological requirements the metabolic processes are presumably raised in individuals who do not put on weight or else a considerable amount of the food is excreted undigested There is no evidence to support the latter view Obesity may be —

1 Developmental This is usually a hereditary condition

2 Nutritional or exogenous This may be due to overeating over drinking alcoholism and lack of exercise In some cases there is water retention in the tissues especially in the fatty tissues Giving up smoking usually results in a gain in weight as the patient eats more Worry and other psychological factors may also result in obesity

3 Metabolic or endogenous This may result from endocrine disturbances such as hypothyroidism, hypothalamic lesions hyper adrenia associated with Cushing's syndrome hyperadreno-corticalism and deficiency of secretion from the ovaries (as at the climacteric) or from the testicles (as in eunuchs)

**Clinical Findings** The weight of the patient is usually several stones above the normal for the age and height the disposition is generally calm and cheerful the only complaint being due to the personal appearance and discomfort of the excess of fat, which may

patient should take 2 tablets of colchicine less than the dose which produced diarrhœa Good results have been obtained by the intramuscular injection of ACTH mg 12 5 every 6 hours for 2 days and then mg 25 daily for 4 to 8 days Cortisone also has a good effect in doses of mg 100 b i d for 3 or 4 days, and then mg 100 daily for 4 to 8 days Colchicine should be given simultaneously in small doses 1 tab (gr 1/240) twice a day providing there is no diarrhœa, and continued for a few days after discontinuing the ACTH or cortisone Butazolidin is also effective in gout in doses of one tab mg 100 every 6 hours for 4 doses then every 8 hours for 3 doses, and then twice daily until the attack subsides The patient should drink plenty of fluids 4 to 8 pints in the 24 hours hot water hot weak tea orangeade or barley water, and he may also have toast milk bread and butter and milk pudding After the acute symptoms have subsided the diet is increased fish and chicken being given Atophan (cincophenum B P ) is a dangerous drug it may cause acute yellow atrophy of the liver and should not be prescribed All infective foci in the mouth should subsequently be eradicated

**Chronic Gout** Gout is considered to be chronic when an attack lasts for several weeks or when attacks recur at short intervals Usually several joints are affected, permanent deformity ensues and the general health is impaired The tophi may ulcerate through the skin and suppuration occur around them (see Fig 66) There may be thickening of the olecranon and prepatellar bursæ The urine may contain a trace of albumin or of glucose Eczema is often present The tophi usually differentiate chronic gout from osteoarthritis although the two conditions may be combined X ray examination of the hands may show punched out areas in the distal parts of the phalanges Fibrositis is sometimes a manifestation of chronic gout

**Treatment** Diet The patient should avoid substances rich in purins such as sweetbreads liver kidneys, brains, sardines anchovies turkey pork veal beef duck goose partridge bacon, rabbit lamb meat soups and extracts, and lentils Purin poor substances are advisable these include white bread, butter cheese eggs rice tapioca milk cereals and green vegetables Fruits such as peaches apples pears grapes cherries, and oranges may be given As the blood uric acid rises on a high fat diet the amount of fat taken should be strictly limited Carbohydrates do not appear to be harmful Plenty of fluid such as hot water should be drunk on rising and retiring Tea coffee and cocoa may be taken in moderation It is better to avoid all alcohol A little dry wine or whisky may be allowed The bowels should be kept open regularly and a saline aperient taken from time to time The continuous use of uricosuric drugs to prevent recurrent attacks of gout and to provoke resolution of tophi is now generally recognised The choice lies between probenecid (Benemid) and salicylates Benemid is put up in 0 5 G tablets Treatment is begun with 1 tab daily then 2 tabs daily and then 3 or 4 tabs daily The maintenance dose is 1 to 4 tabs daily, according to the blood uric acid level Benemid causes a rapid fall in the blood uric acid level from 10 mg /100 ml to a level of

*10 p m* Water biscuits (2) Milk (1 oz) Tea Saccharin for sweetening at all meals

In severe cases of obesity where an adult weighs over 18 st, a diet of 500 calories, containing protein 36 G, fat 16 G and carbohydrate 56 G will be required if weight is to be lost at the rate of 3 to 4 lb a week. When the weight has been reduced by 5 or 6 st the 1 000 calories diet can be substituted

Fluid should be restricted to about 50 oz in the 24 hours Appetite may be reduced by taking amphetamine sulphate mg 5 tablet half an hour before breakfast and lunch In obesity due to hypothyroidism thyroideum gr ¼ to 1 can be given daily and gradually increased

Mersalyl a salt poor diet and restriction of fluid intake to 35 or 40 oz in the 24 hours should be used for excess of weight due to water retention Exercise should also be taken a walk of a mile or more daily or simple exercises such as contracting the trunk and abdominal muscles

### Localised Lipomatosis

Localised deposits of fat may occur in the following conditions — 1 Lipoma single or multiple These should be removed if growing rapidly owing to the risk of sarcomatous changes 2 Localised obesity the fat accumulating over the hips or abdomen Vibration and massage are useful in these cases Gluteal humps of fat are met with in Bushmen and Hottentots They are analagous to the camel s hump and become flabby during starvation The condition is known as steatopygy Symmetrical excess of adipose tissue may form in the neck constituting the fat neck of Madelung 3 Adiposis dolorosa 4 Pseudo hypertrophic muscular dystrophy

### Lipodystrophia Progressiva

A disease of children characterised by progressive wasting of the face neck arms thorax and abdomen The cause is not known it has been suggested that it is due to a tropho neurosis The buttocks and legs are usually normal but in some cases are the only parts affected The wasting is due to loss of fat the tone of the muscles being normal *The loss of fat is steadily progressive for periods up to 2 or 10 years* The general health is not impaired

### Localised Lipodystrophy

Loss of subcutaneous fat occurs in some individuals in the arms or legs in association with repeated injections of insulin (see p 660) Local panatrophy may also occur apart from insulin injections with wasting of the subcutaneous tissues including muscles It may be seen in the arm or leg and the cause is unknown

### Ochronosis

**Definition.** A rare disease characterised by pigmentation of cartilage and skin arthritis and urinary changes

**Etiology** There are three groups of cases 1 Associated with alkaptonuria an inborn error of metabolism of tyrosine and phenylalanine in which homogentisic acid is excreted in the urine 2 Associated with

also cause dyspnœa from cardiac embarrassment Investigation of cases of obesity involves first an inquiry into the history as regards heredity, diet and exercise An estimation should be made of the basal metabolic rate sugar tolerance and water elimination, and special tests performed with respect to the ductless glands Unless the obesity is due to hypothyroidism, the basal metabolic rate is usually within normal limits

**Course and Complications** The condition is often slowly progressive, and complications may occur, *such as flat feet arthritis of the knees* hypertension fatty infiltration or degeneration of the heart diabetes mellitus and chronic bronchitis

**Prognosis** Obesity tends to lower the expectation of life, owing to the liability to complications , further, severe illnesses and abdominal operations are less well borne

**Treatment** *Prophylactic* Where a hereditary tendency exists a regular check should be kept on the weight Any increase should at once be countered by a dietetic restriction or increase in exercise As a man weighing 70 kg will only use up 140 calories on walking 2½ miles in an hour it can be seen that reduction of diet is of greater importance than an increase of exercise in keeping the weight within bounds

*Curative* It is usually difficult to persuade the patient that the only source of his excessive fat is what he puts in his mouth In developed cases of exogenous obesity the weight should be gradually reduced by about 2 to 3 pounds a week The caloric value of the patient s normal *diet should first be determined it will often be found to be high over* 4 000 calories Sugar potatoes cakes sweets, butter jam marmalade honey, puddings pastries ices sausages nuts cheese wines and beer should then be eliminated and bread reduced Weight is sometimes lost more quickly if the patient is kept in bed for a week or two at the begin ning of the treatment If this does not produce sufficient loss in weight the patient should be placed on a special diet containing about 1 000 calories It should contain about 1 G of protein per kg of body weight and not too much fat or severe acidosis is liable to develop The follow ing diet of about 1 000 calories containing approximately protein 80 G , fat 38 G and carbohydrate 77 G may be used

*Breakfast* Grilled fish (4 oz ) or grilled fish (2½ oz ) and egg (1) or bacon (2/3 oz ) and tomatoes (3 oz ) or cold ham (2 oz ) or bacon (½ oz ) and egg (1) or eggs (2) boiled or poached Toast (½ oz ) Butter (⅛ oz ) Sugarless marmalade (½ oz ) Milk (1½ oz ) Tea

*Lunch* Roast beef or mutton hot or cold (2 oz ) or grilled steak, boiled beef grilled chop boiled mutton roast turkey or chicken (2½ oz ) or veal (3 oz ) or grilled sweetbreads (3 oz ) with bacon (½ oz ) or liver (3½ oz ) Green vegetables (4 oz ) or salad (4 oz ) Stewed fruit (3 oz ) or fresh fruit (4 oz ) Toast (½ oz ) Butter (⅛ oz )

*Dinner* Fried fish (2½ oz ) or meats as at lunch, or eggs (2) boiled poached or made into an omelette Vegetables fruit toast and butter as at lunch Toast may be increased to 1 oz if the vegetables are omitted Tea

fusions for aplastic anæmia has been described, and also dietary hæmochromatosis in which there is increased absorption of iron

**Pathology** Hæmosiderin is deposited in excess in nearly all the tissues including the skin Hæmofuscin (iron free) which is related to melanin and contains sulphur is deposited in the heart and intestinal muscle This does not result from increased blood destruction but may be due to inability of the organs to excrete the pigment The liver pancreas and spleen are cirrhotic and the glycosuria is considered to be secondary to the pancreatic changes (chronic interstitial pancreatitis)

**Clinical Findings** The patient is almost invariably an adult over the age of 40 who notices darkening of his skin or else complains of the symptoms of diabetes such as thirst polyuria and loss of weight or of those due to chronic acidosis such as headache and neuritis The late age of onset is due to the gradual accumulation of iron in the tissues

*On Examination* The body is somewhat wasted the skin shows excess of pigmentation especially in the face axillæ and groins and pigment may be deposited in the mouth but there is no jaundice The pigmentation may be ashy grey slaty blue due to iron or bronze due to melanin In some cases cutaneous pigmentation is absent The liver is enlarged firm and irregular and the spleen may be felt There is sexual hypoplasia with loss of pubic and axillary hair The urine in about 80% *of cases contains glucose and acetone bodies may be present Hæmo* siderin may be found in the urinary deposit A blood sugar tolerance test shows the typical diabetic curve when glycosuria is present In the early stages there may be no glycosuria and in some cases there is no cutaneous pigmentation The serum iron level is raised above the normal of 80 to 180 microgrammes per 100 ml

**Differential Diagnosis** Other causes of pigmentation must be excluded such as Addison s disease The presence of iron in the skin and sweat glands is diagnostic of hæmochromatosis The hepatic en largement should be differentiated from cirrhosis due to other causes or to carcinoma Needle biopsy will establish the diagnosis The association of pigmentation enlarged liver and glycosuria is very suggestive

**Course and Complications** The course is usually slowly progressive Ascites may develop Other complications include rupture of œso phageal varices pulmonary tuberculosis and diabetic coma

**Prognosis** This is rather more unfavourable than in the case of diabetes mellitus Death may occur from diabetes from cardiac failure from cirrhosis of the liver or from hepatoma

**Treatment** This is similar to the treatment employed in diabetes mellitus when glycosuria is present Repeated venesections have been suggested such as 500 ml every week These will remove about 250 mg iron from the blood and a corresponding amount passes from the tissues into the blood The results of venesection as a long term treatment have not yet been determined

## Primary Amyloidosis

The cause of this rare condition is unknown The tissues in which amyloid is deposited include the heart the skeletal and smooth muscles

chronic absorption of carbolic acid, used for dressing wounds, usually ulcers on the legs for prolonged periods such as 20 or 30 years Carboluria is usually present 3 Occurring apart from either of these conditions

**Pathology** Melanin is deposited as yellow brown particles in the cartilage of the ears nose eyelids, trachea, bronchi and in the ligaments and fibrous tissues of the body Chronic osteoarthritis may be present in the large joints In alkaptonuria homogentisic acid is passed in the urine owing to incomplete metabolism of tyrosine

**Clinical Findings** The patient is usually an adult who seeks advice either on account of the pigmentation of the ears or face or because of arthritis A child may be brought to the doctor because his urine darkens on standing, or the patient may seek advice because he has been rejected for life assurance In the cases due to carboluria a history is usually obtained of a wound being treated with carbolic dressings for a prolonged period

*On Examination* The cartilaginous parts of the ears appear bluish and a blue black area of pigmentation may be seen in the sclerotics The tendons of the hands are pigmented and the skin of the face and hands may be yellow brown or brownish black in patches Osteo arthritis may be found in the knees or other joints X ray examination may show calcification of the intervertebral discs The urine may contain homogentisic acid becoming dark on standing staining the clothes and reducing Benedict s and Fehling s solutions or it may give the reaction for carboluria

**Differential Diagnosis** Alkaptonuria must be differentiated from glycosuria by chemical tests Yeast does not ferment homogentisic acid

**Course and Complications** Alkaptonuria is a congenital abnormality and persists through life

**Prognosis** Alkaptonuria does not shorten life

**Treatment** *Prophylactic* Carbolic acid should not be used for dressings for prolonged periods

*Curative* If the ochronosis is due to carbolic acid the dressings must be discontinued in alkaptonuria the protein in the diet should not exceed 100 G a day

## Hæmochromatosis

*(Diabète Bronzé)*

**Definition** A disease characterised by pigmentation of the skin, fibrosis of the liver and pancreas and glycosuria

**Etiology** Hæmochromatosis is probably due to an inborn error of iron metabolism Males predominate over females in the proportion of 20 to 1 The fundamental abnormality appears to be an increased absorption of iron possibly due to the greater reducing power of the intestinal reducing cells which form more ferrous iron than normal The diminished incidence in women is probably due to blood losses at menstruation and pregnancy which get rid of excessive iron in the blood Transfusion hæmochromatosis occurring after over 100 trans

# CHAPTER XII

# *THE DUCTLESS GLANDS*

## THE THYROID GLAND

**Introductory** The follicles (vesicles or alveoli) of the thyroid gland contain colloid secreted by the lining cuboidal epithelium The thyroid hormone thyroxine is present in the colloid which is rich in iodine Thyroxine has been obtained from the gland and also synthesised One milligramme of thyroxine is said to raise the B M R of man by 3% A daily intake of 0 16 mg of iodine is sufficient to maintain the thyroid function It is believed that thyroxine is formed from tyrosine combined with iodine Iodine is liberated from iodides in the presence of peroxidase manganese and oxygen Iodine combines with tyrosine in the presence of a cytochrome enzyme to form first mono iodotyrosine and then di iodotyrosine Di iodotyrosine by taking up two atoms of iodine, is converted in the presence of a cytochrome enzyme into thyroxine This is stored in the thyroid as thyroglobulin Thyro globulin is acted on by a proteolytic enzyme and thyroxine is liberated A substance which is about five times as active as thyroxine is also present in the circulation It is tri iodo thyronine and it differs from thyroxine in containing three instead of four iodine atoms in its molecule Iodine exerts its antithyroid effect partly by inactivating the proteolytic enzyme and partly by inactivating the thyrotropic hormone of the anterior pituitary Thiourea exerts its antithyroid effect by preventing the liberation of iodine from iodides and by interfering with the peroxidase system The thyroid secretion is regulated by the thyrotropic hormone secreted by the anterior lobe of the pituitary This hormone stimulates the thyroid to form thyroxine but thyroxine inhibits the pituitary production of the thyrotropic hormone There is thus a balance between the activities of the pituitary and the thyroid The thyrotropic hormone in addition causes increased vascularity and hyperplasia of the thyroid and in some cases exoph thalmos

### Simple Goitre

*(Endemic and Sporadic Goitre Diffuse Parenchymatous Goitre or Colloid Goitre Nodular Goitre with Localised or Multiple Adenomata)*

**Definition** General or local enlargement of the thyroid gland without marked disturbance of thyroid function The enlargement is neither due to inflammation nor to malignant disease

**Etiology** Goitre is produced by the thyrotropic hormone of the pituitary In endemic localities where iodine is deficient in the water babies are born with a goitre due to a diffuse hyperplasia of the gland Iodinated protein inhibits the production of the thyrotropic hormone

the tongue the arteries and the skin The spleen liver kidneys and adrenals are less often involved The tongue is enlarged papules plaques and localised hæmorrhages may be seen in the skin and mucous membranes The disease is met with after the age of 40 years the clinical picture being usually that of congestive heart failure with at times peripheral neuropathy There may be difficulty in speech and in swallowing Biopsy of the tongue or of a skin lesion may establish the diagnosis but the congo red test for amyloid is unreliable Death usually occurs from heart failure in about 3 years from the date of diagnosis

### The Fanconi Syndrome

In this disease there is a complicated assortment of metabolic errors due to a congenital defect in the renal tubules which affects both their secretory and re absorptive functions As a result of a defect in the proximal convoluted tubules there is excessive loss in the urine of glucose phosphate calcium bicarbonate amino acids acetoacetic acid potassium and ammonia The continuous hypophosphatæmia results in rickets in children and in osteomalacia in adults

*Renal tubular acidosis* resembles clinically the Fanconi *syndrome*, but it is less severe There is not however renal glycosuria and no loss of amino acids in the urine

Treatment Sodium bicarbonate should be given by mouth to restore the electrolytic balance and for the softening of bones calcium and vitamin D as in the treatment of rickets

**Differential Diagnosis** There is usually little difficulty in recognising that the swelling is due to the thyroid gland A diffuse swelling must be differentiated from that associated with Graves disease and a localised adenoma is distinguished from a toxic adenoma by the absence of severe symptoms of hyperthyroidism and from a malignant tumour or Riedel s chronic thyroiditis by the absence of marked hardness

**Course and Complications** A simple goitre occurring at puberty or in connection with pregnancy often disappears in a few months If definitely associated with a locality the goitre usually persists throughout the patient s residence there and if fibrosis has occurred it is usually permanent An adenoma may undergo toxic changes a hæmorrhage may occur into it or it may become malignant

**Prognosis** This is favourable and life is not usually shortened

**Treatment** *Prophylactic* On the supposition that the cause is iodine deficiency iodine may be added to drinking water in goitrous districts (1 lb sodium iodide to 50 000 gallons water) and iodised table salt used (1 part pot iod to 5 000 parts salt) or a course of sodium iodide can be given to school children in the spring and autumn gr 3 daily for 10 days This latter method has proved successful in America Overdosage must be avoided owing to the danger of inducing thyrotoxicosis A pure supply of drinking water or the use of boiled water in goitrous districts in India has also produced good results

*Curative* Small doses of iodine are beneficial although not always curative If feasible the basal metabolic rate should be determined and iodine given to those patients whose metabolism is subnormal or normal Thus sodium iodide gr 1 may be given t d s or liq iodi simplex 4 to 10 drops t d s in milk or Lugol s iodine solution (liq iodi aquosus B P ) 6 drops t d s in milk An operation is required to remove an adenoma causing pressure symptoms It may also be performed in cases of disfigurement or to forestall the possibility of malignant changes An adenoma should not be treated with iodine as it may become toxic

## Hyperthyroidism

(*Thyrotoxicosis Exophthalmic Goitre Graves Disease Parry s Disease Basedow s Disease Flajani s Disease*)

**Definition** A disease characterised by overactivity of the thyroid gland with sympathetic disturbances throughout the body

**Etiology** The cause is not known Graves disease is liable to follow infections such as influenza diphtheria sinusitis tonsillitis etc It may be associated with a nervous shock but it was not common amongst soldiers in the 1914–18 or 1939–45 wars It may be due to overactivity of the adrenals and sympathetic stimulation but there is no proof of this Experimentally stimulation of the thyroid in the cat by joining the central end of the cut phrenic nerve to the peripheral end of the divided sympathetic results in tachycardia exophthalmos and increased metabolic rate It is not known whether the primary lesion is in the thyroid gland or elsewhere in the body All the clinical features of

so that if sufficient iodine is available the goitre is converted into a simple or colloid goitre Certain substances are goitrogenic thus rabbits fed on cabbage develop goitres Other goitrogenic substances include foods of the Brassica family including soya beans also thiourea and its derivatives sulphonamides and thiocyanates It is not known whether in man goitre is produced by eating excess of goitrogenous vegetable substances There are two main theories as to the etiology of colloid goitre 1 Deficiency of iodine in the diet or the drinking water 2 Impurity of the water due to organisms or suspended calcareous matter Possibly in goitrous water there is something which prevents the utilisation of iodine, as endemic goitre has been eradicated in some districts by giving pure water free from all iodine or by adding iodine to the water *Predisposing causes* 1 Locality Derbyshire and the Thames Valley in England the Swiss Alps Pyrenees Maritime Alps Himalayas Rocky Mountains etc Air borne iodine is present within three miles of the sea coast 2 Age Children and young adults 3 Sex Chiefly females and often associated with puberty, pregnancy and lactation

**Pathology** *Colloid Goitre* The gland is uniformly enlarged The vesicles are distended with colloid and lined with atrophied epithelium The iodine content is increased

*Parenchymatous Goitre* The gland is uniformly enlarged The vesicles contain less colloid than normal and the epithelium tends to be columnar shaped The iodine is diminished

*Nodular Goitre* Localised or diffuse adenomata may occur The adenoma may develop from a fœtal rest The gland may be smaller than normal with increased fibrous tissue The adenoma may be solid or cystic and hæmorrhages may be present in the gland Pressure of the gland may flatten the trachea from side to side, producing a scabbard effect

**Clinical Findings** The patient is usually a young adult who notices a swelling in the neck There may be aching a feeling of fulness or some dyspnœa There is rarely dysphagia but cough is not uncommon

*On Examination* With a colloid or parenchymatous goitre a diffuse swelling of the thyroid gland is found which moves with deglutition It is not hard but may become firm and irregular in long standing cases owing to fibrosis In nodular goitre single or multiple localised swellings are found which are not very hard and may be firm if enclosed in a fibrous capsule A cystic adenoma feels more round and tense In some cases the enlargement is mainly retrosternal, and can then be diagnosed by X rays but the pressure signs may be obvious such as enlargement of the cervical veins stridor hoarseness (pressure on the recurrent laryngeal nerve) and a small pupil (pressure on the cervical sympathetic) In about 60% of cases of colloid goitre there is evidence either of hyper or hypothyroidism In the former there may be tachycardia nervousness an increased basal metabolic rate and diminished sugar tolerance In the latter there is a tendency to obesity, sluggishness bradycardia, lowered basal metabolic rate and increased sugar tolerance

fingers are seen when the hands are held out 4 Eye signs These include exophthalmos, the prominence of the eyes being generally bilateral rarely unilateral Exophthalmos is present in about 95% of cases Corneal ulceration may occur *Von Graefe s sign* When the patient looks down there is lagging of the upper lid *Stellwag s sign* Blinking is infrequent *Moebius sign* Lack of convergence of the eyes when the patient looks at an object which is brought near to him *Joffroy s sign* When the patient looks up the forehead is not wrinkled *Dalrymple s sign* Wideness of the palpebral fissure due to retraction of the upper lid The Merseburg triad of signs described by Basedow in a patient living in Merseburg consists of goitre exophthalmos and tachycardia The heart The impulse is forcible and the apex may be *displaced a little outwards and downwards There may* be irregularity due to premature systoles or auricular fibrillation with or without congestive heart failure Blood pressure This is raised in about 10% of cases When raised there is an increase of the pulse pressure over the normal average of 40 *e g* systolic 180 and diastolic 100 Localised areas of pre tibial myxœdema (myxœdema circumscriptum thyrotoxicum) are met with in some cases Similar patches may occur on the back of the leg There is mucoid degeneration of the connective tissue The electrocardiogram This is normal in over 80% of cases and flat or inverted T waves are the commonest abnormality The blood Lymphocytosis has been described but is not characteristic The sedimentation rate of the red cells is occasionally raised The sugar tolerance is often diminished although the resting blood sugar may be normal The blood cholesterol is usually low below 140 mg per 100 ml (normal 140 to 280 mg per 100 ml) The average amount of plasma protein bound iodine in the metabolically normal adult is 6 6 microgrammes per 100 ml In thyrotoxicosis the values vary between 8 and 35 microgrammes per 100 ml The percentage uptake of radioiodine after giving by mouth a tracer *dose of 30 to 50 microcuries of* $I^{131}$ *is increased in thyrotoxicosis* The percentage amount in the thyroid gland is estimated by a Geiger Muller counter at 24 hours Normally the gland retains 30 to 40% in hyperthyroidism between 40 to 90% is present in the gland, and in hypo*thyroidism the figure is between* + and − 10% *There is* some overlap in these figures for at times in thyrotoxicosis less than 30% is retained at 24 hours The urine There may be glycosuria after a carbohydrate rich meal but often the renal threshold is raised so that no glycosuria occurs when the blood sugar reaches the normal leak point of 180 mg per 100 ml Creatinuria is often found on a creatine free diet Marked polyuria resembling that of diabetes insipidus is noted in some cases Decalcification of bones is very constantly met with and calcium balance experiments show an increased loss both of calcium and phosphorus in thyrotoxicosis The basal metabolic rate is increased to a varying degree usually between + 20% and + 60% A guide can be obtained by using Read s formula B M R = 0 75 (P R + 0 74 P P) − 72 where P R = the resting pulse rate and P P the resting pulse pressure *i e* the difference between the systolic and diastolic pressures

thyrotoxicosis, except exophthalmos, can be produced by the administration of thyroid or thyroxine *Predisposing causes* 1 Age 15 to 40 Graves' disease has been seen in a newborn infant 2 Sex More common in females 3 Absence of any previous goitre 4 Locality Districts free from endemic goitre 5 Heredity A thyrotoxic diathesis may be transmitted

**Pathology** The thyroid gland is enlarged and vascular with increased fibrous tissue There is an increase in the alveolar epithelium which may project in columnar celled ridges nearly filling the lumen of the vesicles The colloid is diminished and the iodine content of the gland low Hyperplastic lymph nodes are constantly present in the gland Adenomata or cysts may be present Rienhoff has shown that after iodine treatment the vesicles contain more colloid, the lining epithelium becomes flat and the hyperplastic gland reverts to the resting colloid state The thymus is often enlarged and the cervical lymph glands are enlarged The cause of the ocular prominence is uncertain It may be due to retro orbital vascular dilatation to lymphocytic infiltration œdema, or fibrosis of the ocular muscles, or to the action of the pituitary thyrotropic principle There is no evidence that it is due to excess of retro orbital fat to stimulation of Muller's muscle which is a vestigial structure in man or to sympathetic stimulation The pupil is not dilated in Graves disease and exophthalmos may occur when the cervical sympathetic is paralysed due to long standing syringomyelia

**Clinical Findings** The patient sometimes gives a history of a shock or intense worry such as receiving his income tax assessment which is followed by symptoms of nervousness, irritability and disturbed sleep The onset may be quite sudden, the patient noticing that one or both of the eyes is prominent or that the neck suddenly swells Other symptoms include loss of weight lassitude nervousness palpitations shortness of breath, moisture of the skin especially on the palms of the hands, and hair falling out The patient usually feels worse in hot weather, the bowels may be relaxed and attacks of vomiting occur Acute abdominal pain may simulate that produced by appendicitis In some cases there are symptoms of diabetes insipidus presumably due to pituitary disturbance In the early stages there may be menorrhagia which is followed later by amenorrhœa

*On Examination* The patient is usually rather thin and has a characteristic startled appearance owing to prominence of the eyes The skin is moist and may be flushed especially on the face and over the neck and manubrium sterni There may be pigmentation, especially on the face arms and trunk with leucodermic patches Carotid pulsation is seen in the neck The typical signs of Graves' disease are 1 Enlargement of the thyroid gland The gland is moderately soft and uniformly enlarged and if the patient raises up the head while lying down the *swelling largely disappears* *Irregularities* may be caused by a cyst or adenoma A systolic murmur is heard over the lateral lobes of the gland 2 Tachycardia The pulse rate is usually over 120 when the patient is at rest 3 Tremors Fine tremors of the

or less for the physicians Operation may be followed by myxœdema requiring permanent thyroid substitution therapy Other complications such as tetany, recurrent laryngeal palsy and exophthalmic ophthalmoplegia are not unknown Against thiouracil treatment may be urged the liability to agranulocytosis which occurs in approximately 2% of cases and which may develop suddenly at any moment during treatment If the goitre is nodular and causing pressure symptoms thiouracil may cause further enlargement and so should not be used and finally the cosmetic result following the removal of a large goitre cannot be achieved by thiouracil Thiouracil has been largely replaced by propyl thiouracil in doses of mg 100 tab t d s The first effect of the drug is usually seen within 1 or 2 weeks but in some cases no improvement is noted for 4 to 12 weeks especially if iodine has been recently given As soon as the B M R is falling satisfactorily usually in about 4 weeks the propyl thiouracil dosage is reduced to mg 50 three times or twice a day When the B M R has fallen to between + 5 and − 10 a maintenance dose of mg 50 to 100 daily should be given The B M R and white cell count should be repeated monthly until the condition is firmly stabilised then three monthly readings should suffice As a routine measure every case should be treated continuously for 10 months Auricular fibrillation may require the simultaneous administration of quinidine although in some cases normal rhythm is restored by thiouracil alone If there is congestive failure it is wise to give digitalis *Overdosage of propyl thiouracil is shown by increase in the size of the* thyroid by a bloated appearance a feeling of heaviness lassitude or depression or by an undue rise in the blood cholesterol and excessive fall in the B M R If there is any increase of exophthalmos, or if the goitre enlarges considerably gr 1 of dried thyroid should be given daily by mouth to diminish the output of thyrotropic hormone from the pituitary

The most important toxic reaction is agranulocytosis which comes on quite suddenly and may rapidly prove fatal It is most likely to occur between the fourth and eighth weeks of treatment but may appear at any time The patient should be told to stop taking the tablets if he has a sore throat or a temperature and to report to his doctor The treatment for agranulocytosis is described on p 527

*Carbimazole* (*Neo Mercazole*) is preferred by many to propyl *thiouracil It is put up in mg 5 tablets The dose is for mild cases* 1 tab 3 or 4 times a day for average cases 1 tab 6 times a day and for severe cases 1 tab 8 or 9 times a day When the B M R has fallen to normal the dose is ½ to 1 tab daily Side effects include skin rashes nausea and agranulocytosis

*Thyroidectomy* This has been made comparatively safe since the introduction by Plummer of the preliminary iodine treatment Iodine is administered in the form of Lugol s solution (liq iodi aquosus B P Add ) One drop of Lugol s solution = mg 6 iodine Ten drops are given t i d in 1 oz of milk The iodine usually causes a reduction of the pulse rate diminution of nervous symptoms and a fall in the B M.R

The result is a positive or negative figure which is the B M R expressed as a percentage Thus if the answer is + 10 the B M R is + 40%

**Differential Diagnosis** A typical case presents no difficulty In an early case there may be unilateral exophthalmos when Graves' disease has to be differentiated from other causes such as a retro orbital tumour, lateral sinus thrombosis or a general disease such as chloroma or xanthomatosis Here a determination of the B M R is a valuable aid to the clinical findings The normal range is between − 15 and + 5 (Aub de Bois) *If there is doubt whether thyrotoxicosis is present the* B M R should be repeated after two weeks iodine treatment In a case of thyrotoxicosis the B M R will fall with the iodine Exophthalmic ophthalmoplegia is thought to be due to the action of the pituitary thyrotropic hormone It is an example of a localised myopathy associated with thyrotoxicosis past or present Individual movements of the eye especially elevation, are affected rather than individual muscles It may follow thyroidectomy (malignant exophthalmos) and then be associated with myxœdema, or the ophthalmoplegia may antedate the severe exophthalmos Early pulmonary tuberculosis may cause difficulty owing to the similarity in both diseases of such symptoms as loss of weight tachycardia weakness and reflex cough Loss of weight frequency of micturition and glycosuria may suggest diabetes mellitus A toxic adenoma must also be differentiated and milder forms of simple hyperthyroidism

**Course and Complications** The course is usually prolonged with definite remissions and exacerbations of the toxic symptoms for periods up to 10 or 20 years Gastro intestinal crises with severe vomiting and diarrhœa may occur, or thyroid crises in which the pulse suddenly becomes extremely rapid with great exhaustion or delirium Fulminating cases with a rapidly progressive course fever and mania are described Complications include myocardial degeneration heart failure, exophthalmic ophthalmoplegia ulceration of the eye myxœdema and diabetes mellitus In some cases there is acute thyrotoxic myopathy characterised by profound muscular weakness, which responds to iodine treatment Chronic thyrotoxic myopathy is characterised by extreme muscular weakness and is cured by treatment of the thyrotoxicosis Thyrotoxic periodic paralysis is a condition in which thyrotoxicosis is associated with familial periodic paralysis In some cases thyroidectomy has cured the attacks Rarely myasthenia gravis and thyrotoxicosis co exist

**Prognosis** The disease is serious but in many cases there appears a natural tendency to improvement and economic recovery may take place in 3 or 4 years in about half the cases although usually some *exophthalmos* persists Restoration to a full normal life may be expected in about 70 to 80% of cases after a successful operation

**Treatment** *Thiouracil* Since the introduction of thiouracil it has become more difficult to decide between medical and surgical treatment in cases of thyrotoxicosis The mortality rate of cases treated medically or surgically is much the same providing the operation is performed by a *highly skilled thyroid surgeon* about ½ to 1% for the surgeons and ½%

### Toxic Adenoma

(*Secondary Graves' Disease Secondary Thyrotoxicosis*)

**Definition** A disease characterised by the presence of one or more adenomata in the thyroid gland later followed by thyrotoxic symptoms

**Etiology** The proliferation of the adenoma may be due to deficiency of iodine

**Pathology** Proliferation of the lining epithelium of the adenoma does not usually occur The follicles contain colloid or the adenoma may be filled with spheroidal cells conforming to the fœtal type

*Clinical Findings The patient is usually an adult over the age of* 35 who has had a goitre for some years Symptoms of hyperthyroidism then appear insidiously

*On Examination* The noteworthy features are the presence of an adenoma in the thyroid the absence of exophthalmos and acute nerve *crises and the liability to auricular fibrillation* The other signs are as for Graves disease but the eye signs are not present It should be noted that the supporters of the view that primary and secondary thyrotoxicosis are one and the same disease state that exophthalmos is rare in toxic adenoma owing to the later age incidence of the onset

**Differential Diagnosis** There is sometimes difficulty in distinguishing secondary from primary Graves disease If the adenoma is small or retrosternal it may not be palpable and a clear cut history *of goitre antedating by several years the thyrotoxic symptoms will not* be obtained

**Course and Complications** The course is typically progressive without remissions Myocardial degeneration is common in untreated cases

**Prognosis** This depends upon the treatment If recognised early and treated adequately there is good hope of permanent recovery

**Treatment** This is as for Graves disease but an operation is indicated in the majority of cases especially when the gland is producing *pressure effects and also owing to the possibility of malignancy*

## Hypothyroidism

### Cretinism

(*Primary or Congenital Hypothyroidism*)

**Definition** A disease resulting from congenital deficiency of thyroid secretion

*Etiology The cause is unknown Cretinism may be associated with* deficiency of iodine in the diet of the mother during pregnancy It is more common in goitrous districts (endemic cretinism) the parents being goitrous but it also occurs sporadically especially in England when the parents rarely have a goitre The sex incidence is about equal

**Pathology** The thyroid gland may be absent (athyreosis) or small and undeveloped and show atrophy of the vesicular epithelium with overgrowth of the connective tissue Adenomata containing little colloid *may be present in the gland of endemic cretins*

**Clinical Findings** The cretin does not develop normally from

The effect is only temporary, and after two weeks or so the condition deteriorates with a rise in the B M R The operation should be performed as soon as the iodine has produced its maximum effect, usually in 10 to 14 days Propyl thiouracil or Neo Mercazole, given as recommended above to reduce the toxicity of the goitre, combined with iodine for 5 days before operation, is preferred by some surgeons and is especially valuable in severe cases of Graves' disease, which do not respond to iodine therapy alone

Should a thyrotoxic crisis occur after the operation, intravenous iodine should be administered in the form of Lugol s solution m 10 in 20 ml of normal saline containing 10% of dextrose This should be injected intravenously every 3 hours until the pulse rate falls In the average case, which has been successfully operated on, work should not be resumed for at least 2 months

*Radioiodine* This treatment is becoming more and more popular, but facilities for its administration are not always available A tracer dose of 30 to 50 microcuries of $I^{131}$ is given by mouth and the per centage uptake in 24 hours is determined From this figure and the estimated weight of the thyroid the therapeutic dose is calculated This is usually about 3 millicuries by mouth If the thyrotoxicosis is not cured in 3 months the dose can be repeated Special precautions have to be taken when radioactive substances are administered There is no danger of producing sterility Whether or not carcinoma of the thyroid may develop after an interval of many years is not yet known

In some cases of severe Graves' disease maniacal symptoms occur For these hyoscin hydrobrom gr 1/200 should be injected subcutaneously and Lugol s solution given in doses up to 100 drops in the 24 hours Intravenous iodine injections as detailed above for the treatment of a thyrotoxic crisis should be administered if the patient is vomiting If the vomiting is severe rectal salines should also be given, preferably by the continuous drip method using 5% dextrose in normal saline

X ray treatment to the thyroid gland is rarely of value and in severe cases sudden death may occur

## Masked Hyperthyroidism

There are some cases of cardio vascular disturbance due to hyper thyroidism in which no enlargement of the thyroid gland can be detected and in which there are no obvious eye signs The patients are usually over 40 The positive features suggesting the diagnosis are Palpitations auricular fibrillation either paroxysmal or continuous especially if resistant to digitalis or quinidine treatment cutaneous vasodilatation loss of weight sweating pigmentation a tendency to diarrhœa and nervousness and a slight degree of staring of the eyes The diagnosis is confirmed by finding a raised B M R In some cases the B M R is only slightly raised or is thought to be normal but after 14 days' iodine treatment it falls, showing that it was above the patient s normal reading

Treatment This is as for Graves disease

## Myxœdema

### (Secondary or Acquired Hypothyroidism)

**Definition** A disease due to acquired deficiency of thyroid secretion

**Etiology** The cause is unknown except in cases resulting from too extensive removal of the gland (cachexia strumipriva) those due to hypopituitarism, or occurring as a sequel of lymphadenoid goitre *Resorcinol ointment applied to varicose ulcers of the legs may act as an* antithyroid agent and cause myxœdema A similar effect may be produced by other antithyroid substances such as thiouracil and radioiodine *Predisposing causes* 1 Age Usually between 30 and 60 2 Sex More common in women 3 Heredity Myxœdema may run in families

**Pathology** The thyroid gland is small. The vesicles are few in number and fibrous tissue is present In some instances there is a colloid goitre The skin shows an increase of subcutaneous connective or fatty tissue but there is no œdema and mucin is not present

**Clinical Findings** The patient is usually an adult over the age of 40 who notices the gradual onset of such symptoms as lack of energy torpor increase in weight deafness supra clavicular swelling loss of hair dryness of the skin sensibility to cold and constipation There may also be pains in the muscles of the arms or legs near the joints stiffness and swelling of the fingers and a painful swelling may occur in a large joint such as the knee The patient feels better in hot weather A few cases with an acute onset have been recorded

*On Examination* The face is somewhat round and expressionless the skin has a yellowish tinge but is red over the malar bones The eyelids may be swollen and so semi-closed The lips may be swollen and the voice guttural The hair is dry and rather scanty and the outer parts of the eyebrows are deficient The tongue may be enlarged and speech and cerebration slow The thyroid gland is usually small the skin is dry the hands are rather clumsy and although the arms and legs appear swollen there is no pitting on pressure Supra-clavicular pads of thickened subcutaneous tissue are characteristic The heart may be dilated and the arteries thickened but the blood pressure is not usually low and may be high The pulse is slow the temperature subnormal the basal metabolic rate is reduced to about $-30\%$ and the sugar tolerance increased The blood cholesterol is raised and varies inversely with the B M R The plasma protein bound iodine is low and the radioiodine uptake is also low (see p 683) A macrocytic or micro cytic anæmia may occur The urine may contain a trace of albumin Menstruation may be irregular if the disease occurs before the climacteric

**Differential Diagnosis** The appearances of a typical case are characteristic Obesity due to other causes must be excluded The patient s relatives may think she is suffering from nerves or that she is a *malade imaginaire* The absence of true œdema differentiates the swelling of the legs from that caused by cardiac or renal disease Swelling of the large joints may suggest osteoarthritis due to other causes and swelling of the hands has been mistaken for rheumatoid arthritis

birth The features which attract attention are delay in growth, intelligence and movement The teeth may erupt late, and constipation is often troublesome

*On Examination* A typical cretin presents a somewhat bloated appearance with lack of expression The hair is scanty dry and brittle the individual hairs appearing to be set too widely apart There may be a coarse down on the trunk the forehead is low the skin thick and dry the nose flat the tongue may be large and the hands are podgy Pads of fat may be felt above the clavicles The abdomen is prominent, and an umbilical hernia may be present The thyroid gland may be difficult to feel or may be enlarged The intelligence is of a very low grade the child being practically an idiot, but quiet and easily managed The pulse is slow the temperature subnormal and the basal metabolic rate low (such as — 40%) X ray examination may reveal delay in ossification of the bones but there is no overgrowth of the epiphyseal cartilage

**Differential Diagnosis** In infancy the condition must be diagnosed from Mongolism congenital or acquired mental deficiency, such as follows encephalitis lethargica or other varieties of dwarfism In Mongolism the appearances are characteristic With congenital mental deficiency the baby may appear normal but difficulty may be noticed in swallowing solid food or in sitting up or there may be a squint In dementia following encephalitis lethargica the child is often excitable, resembling an animal

**Course and Complications** The course depends upon efficient treatment and the date of its commencement An untreated cretin becomes an unintelligent dwarf who is often a deaf mute and lacks sexual power If not treated for some years but little improvement can be expected, especially as regards mentality Complications are usually due to intercurrent disease

**Prognosis** This is good if the condition is recognised and treated early *but thyroid extract will probably be needed during* the whole of life and the mentality is usually but not invariably subnormal The response to thyroid treatment is not so good in endemic cases

**Treatment** *Prophylactic* In goitrous districts expectant mothers should be given sod iod gr 1 t d s

*Curative* The initial dose of thyroid extract for a cretin should always be small such as thyroideum gr ¼, increasing to gr 1 or 2 t d s, according to the age of the child and the response obtained Sufferers from thyroid deficiency are much more susceptible to thyroid extract than are *normal individuals Thyroid treatment should* be discontinued temporarily during any intercurrent infection The child should be weighed every week An operation may be required to remove an adenoma causing pressure

Toxic symptoms due to overdosage include tachycardia sweating, vomiting diarrhœa, restlessness and loss of weight Should these appear the thyroid extract must be stopped and when the symptoms have passed off, the treatment should be started again with a smaller dose

inflammatory one with fibrous tissue formation possibly secondary to perithyroiditis The fibrous tissue in the affected portion of the gland invades surrounding structures such as the trachea and cervical muscles and the carotid sheath Men and women are equally affected and it may occur in young people There are often symptoms due to pressure on the trachea œsophagus or recurrent laryngeal nerve and pain may be referred to the ear These symptoms may be out of proportion to the size of the tumour The skin does not adhere to the swelling Owing to the difficulty in distinguishing it from a malignant tumour, removal by operation is advisable but complete removal may be impossible on account of its adhesion to surrounding structures

### Lymphadenoid Goitre

The cause of this condition first described by Hashimoto is unknown Women over the age of 45 are chiefly affected The thyroid is firm and infiltrated with lymphocytes and the normal colloid is absent The gland is uniformly enlarged and various pressure effects may be noticed Myxœdema may ensue Treatment consists in the administration of thyroid extract for the deficiency symptoms and operative removal to relieve pressure effects

### Tumours of the Thyroid

Simple tumours include an adenoma a fibroma and a papilloma which are rare A teratoma is uncommon and gumma extremely rare A parathyroid tumour may be situated in the thyroid and resemble clinically a thyroid adenoma Carcinoma is not very uncommon It is usually a primary growth but may be secondary to a tumour in the breast tongue or stomach or the gland may be involved by a direct spread from carcinoma of the œsophagus The characteristic features are the stony hardness of the tumour its tendency to adhere to the skin trachea or larynx and the pressure symptoms due to deviation of the trachea compression of the œsophagus or involvement of adjacent nerves, such as the recurrent laryngeal and sympathetic Pain may be felt in the ear Secondary deposits are very liable to form in glands the lungs bones the liver the eyes and in the skin The diagnosis is often very difficult but malignancy should be suspected if a tumour grows rapidly after a period of inactivity if it becomes very hard and if in addition toxic symptoms develop The early onset of dysphagia is also very suggestive Treatment is surgical followed by the administration of radioiodine but the prognosis is usually grave A secondary deposit in bone can give rise to hyperthyroidism after thyroidectomy Its removal may lead to myxœdema In about 10% of metastases there is a favourable response to the oral administration of a large dose of $I^{131}$ such as 200 millicuries Sarcoma of the thyroid is very rare it is usually a round celled rapidly growing tumour

### Granulomata and Cysts

Miliary tuberculosis may affect the thyroid and in secondary syphilis the gland may enlarge or a gumma may form later A hydatid

Some cases have been mistaken for essential hypertension Paralytic ileus has been described in severe hypothyroidism in adults The "forme fruste" of essential xanthomatosis must be distinguished from myxœdema In the former the B M R is low the blood cholesterol raised and the patient complains of being easily fatigued The skin is smooth and the hair is fine, and the patient is mentally and physically alert Treatment consists in restricting the cholesterol in diet

**Course and Complications** The condition is usually slowly progressive but arrest may occur at any stage apart from treatment There may be marked mental deterioration, even the so called "myxœdematous madness" Complications are due to intercurrent infections Death from hypothermic coma has been described the condition not responding to thyroid treatment

**Prognosis** There is usually marked improvement with treatment

**Treatment** Thyroideum should be given, beginning with gr ¼ and increasing gradually to gr 2 or more t d s until the condition is relieved when the minimal dose required to maintain the improvement must be worked out Care must be taken to give small doses or palpitations angina or cardiac failure may ensue The return of the blood cholesterol to normal usually indicates that a sufficiency of thyroid is being given although the B M R may still be below normal The urinary output is usually increased by the thyroid treatment Constipation should be *treated by attention to the diet, and the use of laxatives if necessary*

## Masked Hypothyroidism in Adults

In masked hypothyroidism the patient presents no clinical features of myxœdema but the B M R is low and the symptoms are relieved by thyroid extract Examples are afforded by certain cases of angina of effort secondary amenorrhœa and dermatomyositis

## Acute Thyroiditis

(*Guervain's Thyroiditis*)

Acute thyroiditis is uncommon, and is more likely to affect a patient who has a goitre than one whose thyroid gland is normal Young people are chiefly affected It may complicate influenza scarlet fever typhoid fever puerperal septicæmia etc, or occur apparently spontaneously The thyroid often swells suddenly and there is pain, dyspnœa and dysphagia There is some fever and at times there are rigors Unless abscesses form the swelling usually disappears in a few days Cold applications should be applied to the neck and sedatives such as pot brom gr 10 to 15 given t i d Propyl thiouracil should be administered as for thyrotoxicosis (see p 685)

## Riedel's Disease

(*Eisenharte Strumitis*)

A portion of the thyroid gland becomes extremely hard like iron, suggesting a malignant tumour The surface of the gland however is smooth and lymphatic glands are not involved The change is a chronic

be low although the total serum calcium is normal 2 Alkalosis This does not result in a decrease in the serum calcium but it may lower the amount of ionised calcium This has not been proved and there is experimental evidence against it Other explanations are that the *alkalosis increases the excitability of nerve fibres or nerve centres, or* limits the available supply of oxygen to the muscles by altering the dissociation curve of oxyhæmoglobin

Tetany may result from 1 *Parathyroid Deficiency* The blood calcium is low It may result from operations on the thyroid gland (tetania parathyreopriva) It also occurs in Graves' disease where the parathyroids may be involved and in epidemics possibly due to para thyroiditis 2 *Deficient Absorption of Calcium* Infantile tetany associated with rickets results from deficient absorption of calcium owing to lack of vitamin D Osteomalacia in adults may be due to a similar cause Tetany may also complicate cœliac disease sprue dysentery cholera and Hirschsprung s disease 3 *Alkalosis* Gastric causes include pyloric stenosis and the administration of large doses of certain alkalis in the treatment of peptic ulcer It has been noted in chronic arsenic poisoning in which there is prolonged vomiting It may also occur in high intestinal obstruction and nephritis Hyperpnœa is a comparatively common cause of tetany It may be hysterical voluntary or result from excessive exercise The serum phosphorus is found to be low if the blood is taken immediately after an attack of hyperventilation tetany In very hot and humid climates hyperpnœic tetany may occur and fireman s cramp in some cases is probably due to alkalosis in others to loss of chlorides

Tetany is said also to occur as the result of certain poisons such as chloroform lead and morphine and to be associated with some nervous diseases such as syringomyelia or cerebral tumours The explanation of these conditions is obscure unless there is hyperventilation It may also be associated with pregnancy and lactation possibly due to exces sive demands for calcium and to deficient absorption of calcium as in osteomalacia

**Pathology** Lesions may be found in the parathyroids such as inflammation hæmorrhage fibrosis miliary tubercles leukæmic *infiltration and a tumour or there may be no lesion present The spasms* affect chiefly small voluntary muscles it is not known whether they are neurogenous or myogenous in origin but certainly both motor and sensory nerves are hyperexcitable It is doubtful if involuntary muscles are affected Cataract may be due to deposition of calcium phosphate in the lens and calcification of cerebral arteries may be a cause of convulsions

**Clinical Findings** Infantile tetany occurs in rickety children between the ages of 6 months and 2 years they often have diarrhœa The tetany may show itself by attacks of laryngo spasm or by carpo pedal spasm or by general convulsions A case of post operative tetany of mild degree illustrates well the condition seen in adults The patient complains of attacks of tingling or numbness in the fingers with stiffness in the fingers or arms and then contractions of the hands the thumb

cyst may also develop in the gland The cystic adenoma has been described on p 680 A degeneration cyst is due to degeneration of an adenoma Hæmorrhage may occur into it Retention cysts are also described

## THE PARATHYROID GLANDS

**Introductory** There are usually four parathyroid glands, situated in close relationship to the middle third and lower poles of the posterior surface of the lateral lobes of the thyroid gland Small accessory glands may be present in the upper or lower pole of the thymus or in the carotid sheath Parathormone, the parathyroid hormone regulates the renal excretion of phosphates An increase in the hormone causes a diminished absorption of phosphate from the glomerular filtrate as it passes through the renal tubules The lowering of phosphate in the blood leads to increased elimination of phosphate, and with it calcium, from the bones Vitamin D has the reverse effect on phosphate, increasing its reabsorption through the renal tubules and increasing deposition of calcium at the epiphyseal ends of the bones In larger doses it causes hypercalcæmia Parathyroidectomy in mammals causes tetany as the result of the fall in the blood calcium

### Hyperparathyroidism

*Acute hyperparathyroidism* is rare The symptoms resemble those resulting from the injection of parathormone into the dog It may result from the accidental over administration of parathormone or be due to a parathyroid adenoma The chief features are anorexia nausea vomiting drowsiness constipation and low blood pressure The blood urea and calcium are raised The condition is liable to be mistaken for uræmia *Chronic hyperparathyroidism* results in generalised osteitis fibrosa

### Hypoparathyroidism

Deficient activity of the parathyroids results in tetany The relationship of tetany to the parathyroid function is not clear in all cases

### Tetany

(*Spasmophilia*)

**Definition** A symptom complex characterised by over excitability of portions of the nervous system with resultant muscular spasm

**Etiology** There are two main causes —

1 Deficiency of calcium in the blood In many cases of tetany the blood calcium is low injection of parathormone or the administration of calcium by mouth intramuscularly or intravenously raises the blood calcium and relieves the spasms of tetany Calcium occurs in the blood in three forms 50% is in organic combination with plasma protein and 50% is present either in un ionised inorganic form or as calcium ions Tetany may be due to a deficiency of calcium ions In some cases the blood calcium figure is low in others the ionised calcium may

does not however always lose consciousness and there is much pain Tetanus and strychnine poisoning are usually easily differentiated Between the spasms the special signs described above are of value Hysterical spasms are not usually bilateral

**Course and Complications** *Recurrence is very liable to occur, the* attacks may last a few minutes or persist for hours or days Cataract is an important complication

**Prognosis** Tetany is a serious condition In infants it may prove fatal with laryngospasm or generalised convulsions, and in adults it is especially serious in association with childbirth gastric dilatation nephritis or after operation on the thyroid

**Treatment** *Prophylactic* In infants prophylactic measures are to be directed to the prevention of rickets

*Curative* The spasms if violent may be controlled by administration of chloroform as a temporary measure of urgency Morphine *should never be given If the blood calcium is below normal a diet* rich in calcium such as eggs milk and green vegetables should be given The optimum daily intake of calcium for a child is 1 G and for an *adult 0 75 G A quart of milk contains 1 G of calcium Calcium* should also be administered as calcium lactate gr 5 to gr 240 t d s by mouth or injected intravenously as calcium gluconate 10 ml of a 10% solution daily until the symptoms are relieved When calcium is given by mouth vitamin D should also be given such as liq calciferol m 20 (4 000 i u ) t i d daily High potency Ostelin tablets containing 50 000 i u may be given 2 tabs daily for 5 days a week without additional calcium Even when the blood calcium is below 6 mg per 100 ml it is seldom necessary to give intramuscular injections of parathormone A fraction of irradiated ergosterol dihydrotachysterol or A T 10 (anti tetanic substance No 10) which does not contain vitamin D when taken by mouth raises the blood calcium It is put up as an oily solution in bottles of 15 ml Three to 10 ml are taken daily for 3 days followed by a weekly maintenance dose of 1 to 7 ml but blood calcium estimations are essential as hypercalcæmia is liable to occur Parathyroid extract by mouth has no effect in raising the blood calcium as the active principle is destroyed in the stomach Cataract requires treatment by operation

Constipation should be relieved by an enema and a colonic washout given every other day with 1 to 2 pints of normal saline until the muscular excitability is normal Sedatives such as chloral hydrate gr 5 to 10 t d s by mouth for an adult may be necessary for the first 2 or 3 days If there is gastric dilatation a tube should be passed and gastric lavage performed with normal saline When the calcium in the blood has been restored to normal the patient should be kept on a diet rich in milk and eggs and calcium lactate *gr 5 to 10* given daily Spontaneous hyperventilation tetany can be terminated by breathing air containing excess of $CO_2$ Tissue acidosis resulting from muscular activity may be the cause of spontaneous termination of hyperventilation tetany In other cases of tetany due to alkalosis the treatment is as described on p 669

being pressed across the palm causing pain The feet may also be affected becoming stiff, the ankles turning in and the toes being flexed Twitching may be noticed in various muscles of the body, and the face and the lips may feel tight during an attack causing difficulty in speech or swallowing The attacks may come on suddenly and be provoked by raising the arms or lying still in a fixed position The patient may also have convulsive fits with or without loss of conscious ness Vision may periodically become dim and finally be lost owing to cataract

*On Examination* During an attack carpo pedal spasm is typical The hands are fixed in the accoucheur s position, with the thumb flexed across the concave palm, fingers adducted and flexed at the metacarpo phalangeal joint, and extended at the interphalangeal joints The wrists and elbows may be flexed The feet The ankles are dorsi flexed and may be inverted with toes flexed and the sole concave The knees are extended The feet and hands may be congested The corners of the mouth may be depressed and the lips protruded (carp mouth) Spasm may also affect the abdominal muscles, or diaphragm and intercostal muscles, causing expiratory apnœa, and lateral deviation of the eyes may occur In a long standing case the skin is dry the nails brittle the teeth become carious and the hair falls out Cataract may develop, and in its early stages can be detected by seeing opacities with the slit-lamp In severe cases general convulsions may occur in adults The increased irritability of the neuro muscular system in latent tetany may be demonstrated by the following signs *Chvostek s sign* A tap over the facial nerve in front of the lobe of the ear provokes varying degrees of contraction of the muscles supplied such as twitching of the angle of the mouth of the outer canthus of the eye cheek forehead and alæ nasi *Trousseau's sign* Compression of the upper arm by a sphygmomanometer armlet with a pressure sufficient to obliterate the radial pulse provokes spasm of the hand in 1 to 5 minutes This may not be present in a well developed case *Schultze s sign* A localised dimpling occurs on tapping the protruded tongue with a patella hammer *Pool's sign* Extension of the brachial plexus by forcible abduction of the arm causes spasm in the hand and arm *Schlesinger s sign* Flexion of the hip with the knee extended causes spasm of the leg *Erb s sign* There is increased excitability of motor nerves to galvanic stimulation A kathodal opening contraction will occur with a current of less than 5 milliampères applied to such a nerve as the external peroneal *Hoffman s sign* Stimulation (mechanical or electrical) of a sensory nerve will cause a muscular spasm In post operative tetany the serum calcium is usually below 7 mg per 100 ml As the calcium falls the serum phosphorus rises In hyperventilation tetany the blood calcium is normal but the phosphorus is low

**Differential Diagnosis** In infants other causes of convulsions must be considered (see p 309) Laryngospasm must be differentiated from other causes of laryngeal obstruction (see p 130) Carpo pedal spasm occurs only in tetany and is diagnostic In adults generalised con vulsions due to tetany have been mistaken for epilepsy the patient

The internal secretion of the medulla is adrenaline and it is inert on oral administration Noradrenaline is also produced in the medulla It lacks the N methyl group of adrenaline When noradrenaline is given intravenously in normal saline it causes a rise of blood pressure, *without disturbing the action of the heart Adrenaline acts on the* junctional tissue between the sympathetic nerve terminations and the cells they activate The results produced by adrenaline injection are similar to those caused by sympathetic stimulation Adrenaline is poured forth into the blood in times of stress The effects produced include dilatation of the pupils rise of blood pressure acceleration of the heart erection of hairs dilatation of bronchioles transference of glycogen from muscles to the liver increase in blood sugar and blood lactic acid relaxation of intestinal muscles and constriction of the intestinal sphincters and of the arterioles of the skin and splanchnic region In times of stress also it is thought that adrenaline stimulates the anterior pituitary by way of the hypothalamus This results in the production of ACTH which causes the adrenal cortex to secrete 11 and 17 oxysteroids A fall in the circulating eosinophils is then produced

## Hyperadrenia

Hypertrophy and tumours may result in overactivity of the supra renals

*The Cortical Tumours* These include an adenoma adeno carcinoma and carcinoma *An adrenal carcinoma often involves the kidney* and metastases occur in abdominal and mediastinal glands and in the lungs liver and brain The tumour may be functionally inert causing pressure effects or physiologically active resulting in virilism or feminism With the physiologically active tumours diagnosis is possible before metastases have occurred and removal of the tumour is often successful The condition is known as hypercortico adrenalism or the adreno genital syndrome

*The Adreno genital Syndrome* This may affect children or adults due to adrenal cortical hyperplasia or to a simple or slowly growing malignant tumour In boys the symptoms include excessive muscular development a low voice and premature sexual development (the infant Hercules) Girls assume male characteristics The onset of menstrua tion is delayed the skin of the face is coarse and red the voice harsh the pubic hair increases there is obesity and the clitoris may enlarge In adults the symptoms and signs closely resemble those of Cushing s syndrome which is probably due to a primary hyperplasia of the adrenal cortex (see p 640) There is a similar distribution of obesity facial hirsuties loss of sexual desire and in women amenorrhœa There may be a persistent glycosuria and high blood pressure *A direct* X *ray* examination of the renal region and one taken after the injection of Pyelectan may suggest the presence of a suprarenal tumour X ray examination after perirenal insufflation of air may show up small adrenal cortical tumours and phæochromocytomata *The diagnosis is aided by* examination of the urine for the presence of the male sex hormone The normal secretion of androgens is 5 to 15 mg sterone a day

### Idiopathic Hypoparathyroidism and Pseudo-Hypoparathyroidism

These are rare conditions In *idiopathic hypoparathyroidism* the parathyroids are fatty and the parathormone secretion is deficient The disease may first manifest itself with epileptiform convulsions or with dementia In addition there is tetany a low serum calcium and a high serum inorganic phosphate There is often thickening of the skin scanty hair, cataract calcification of the basal ganglia of the brain and thickening of the cortex of the long bones

In *pseudo hypoparathyroidism* the parathyroids are normal or hyperplastic but the tissues are resistant to the parathormone secretion The patient is often short of stature with a round face and presents the other features of hypoparathyroidism The treatment for the two conditions is the same the administration of dihydrotachysterol or of calciferol

## THE SUPRARENAL GLANDS

**Introductory** The suprarenal gland is composed of two parts a cortex and medulla, which in elasmobranch fishes remain separate , the cortex forming an inter renal body Further the carotid body the para ganglionic bodies and accessory suprarenals belong to the same system The cortex is derived from mesoderm and is composed of cells containing lipoid granules The medulla is of sympathetic nervous origin and contains chromophil cells around which preganglionic branches of the splanchnic nerves end The cortex of the glands is necessary to life Watery extracts of the adrenal cortex contain a relatively small amount of the cortical hormones but possess to some degree both carbohydrate and sodium regulating properties Such a preparation is Eucortone 1 ml of which is equivalent to 75 G of the adrenal cortex About 30 crystalline steroid substances have been isolated from the cortex of which 6 are physiologically active The hormone present in greatest amount is 17 hydroxycorticosterone or compound F of Kendall Cortisone compound E of Kendall is 17 hydroxy 11 dehydro corticosterone Other active steroids include 11 desoxycorticosterone corticosterone 11 dehydrocorticosterone and aldosterone Cortisone has been synthesised from bile acids and other sources (see p 640) and desoxycorticosterone has also been synthesised and is available in the form of DOCA.

These active hormones may be classified in three groups—whether they are all derived from a common ancestor is not known

1 *Mineralocorticoids* These include deoxycortone acetate (B P ) (DOCA) and aldosterone (electrocortin) They lead to a retention of sodium chloride and water and a loss of potassium

2 *Glucocorticoids* These are concerned with gluconeogenesis They increase protein and fat utilisation and spare carbohydrates Compound F or Compound F material causes a conversion of protein to carbohydrate

3 *Androgenic substances* (nitrogen retention hormones) These are concerned with protein anabolism with the growth of pubic and axillary hair and with the maturation of bone marrow

in the liver (Pepper syndrome) and with the left suprarenal the bones and left eye may be affected (Hutchison syndrome)

In Graves disease or after shock overactivity of the suprarenals may be present with diminished sugar tolerance and glycosuria, the sugar being derived from glycogen in the liver and muscles and possibly from protein At the climacteric some of the symptoms such as rise of blood pressure may be due to hyperadrenia

### Hypoadrenia

The most important manifestation of hypoadrenia is Addison's disease Other conditions which may be associated with hypoadrenia are —Circulatory failure and shock symptoms as in diphtheria typhoid fever scarlet fever and cholera Hæmorrhagic necrosis of the supra renals may be the lesion in these cases Acute suprarenal hæmorrhage involving the medulla may occur in the fulminating type of meningo coccal septicæmia associated with a petechial rash This is usually rapidly fatal and is known as the Waterhouse Friderichsen syndrome In congenital hydrocephalus the medulla of the suprarenals has been found absent in some cases Progeria may be due to a hypoplasia of the cortex

### Addison s Disease

**Definition** A disease characterised by low blood pressure gastro-intestinal disturbances weakness and pigmentation of the skin

**Etiology** Addison s disease is caused by a lesion of the suprarenals and less commonly of the splanchnic ganglia It is rather more frequent in males

**Pathology** Addison in 1855 based his description on 11 cases of these the suprarenals were tuberculous in 5 carcinomatous in 3 fibrotic in 2 and an acute hæmorrhage had occurred in 1 In 4 of his cases only 1 gland was affected The glands may also be gummatous atrophic suppurative or affected by Hodgkin s disease or by a diffuse reticulo-endotheliosis or they may be normal but the splanchnic ganglia are then diseased

**Clinical Findings** The patient is usually an adult between the ages of 20 and 40 who notices progressive weakness loss of appetite wasting darkening of the skin and coldness of the extremities Pigmentation may precede the other symptoms of Addison s disease by several years Vomiting may occur from time to time or be very persistent and there may be diarrhœa Rarely the disease has an acute onset

*On Examination* The patient appears somewhat wasted There is chestnut brown pigmentation of the skin especially on the face the backs of the hands axillæ round the nipples and navel in the pubic region and where there is pressure as at the waist or garter line Small black spots may be seen especially on the forearms leucodermic patches may also occur and the hairs growing from them may be blanched Slaty pigmentation may occur on the tongue the buccal mucous membrane or in the conjunctivæ anus or vagina The blood pressure is low usually below 100 mm Hg systolic The pulse is

Female patients with a cortical tumour often excrete over 100 mg "sterone," and with male patients a moderately high androgen excretion is found The excretion of the 17 ketosteroids is usually increased to 4 to 20 times the normal, whereas it is usually diminished in Cushing s syndrome The 17 ketosteroids are derived from the breakdown products of testosterone They are excreted in the urine The daily excretion is normally greater in men (3 5 to 15 mg ) than in women (1 7 to 12 6 mg ) In women ovariectomy has little or no effect on the *amount excreted but in Addison s disease* (cortical insufficiency) the yield drops to zero Therefore androgens in women arise chiefly in the adrenal cortex In males the testes and the adrenal cortex both probably contribute to the androgen fraction A distinction between adrenal cortical hyperplasia and adrenal cortical tumour can be made by the intravenous injection of mg 50 to 100 of hydrocortisone during 24 hours With hyperplasia there is a rapid fall in 17 ketosteroid excretion in the urine, which does not occur with a cortical tumour

Adrenal cortical hyperplasia responds to the intramuscular injection of cortisone in doses of mg 25 daily The maintenance dose should be checked by estimation of the ketosteroid excretion in the urine which should be maintained at about *mg* 8 daily

*Conn s Syndrome* (primary hyperaldosteronism) This is due to an adrenocortical tumour usually an adenoma which is secreting an excessive amount of aldosterone The patient suffers from muscular weakness or paralysis cramps limb pains hypertension and polyuria There is retention of sodium with a low blood potassium level and excessive excretion of potassium in the urine

*The Medullary Tumours* The *phæochromocytoma* This tumour arises in the chromaffin tissue of the medulla or in the paraganglia of the sympathetic nervous system It may result in paroxysms of high blood pressure with severe headache nausea vomiting shivering sweating pallor tachycardia and fever In other cases the blood pressure is continuously raised Certain tests have been introduced to substantiate the diagnosis In cases with sustained hypertension mg 5 of Rogitine (phentolamine) may be injected intravenously The patient must have no sedative for 24 hours before the test and must lie quietly for half an hour before the test is made The blood pressure is recorded on the other arm every half minute for 10 minutes In an actively secreting tumour the systolic blood pressure falls by about 60 mm Hg and the diastolic pressure by about 25 mm Hg If the blood pressure falls too low Methedrine mg 30 should be injected intravenously With a phæochromocytoma there is usually an increased excretion of noradrenaline in the urine Thus whereas the normal amount of pressor *amines in the 24 hour excretion of urine is* 50 micrograms with a phæochromocytoma the excretion may rise to 100 micrograms or more Treatment consists in removal of the tumour special precautions being taken to combat the severe fall in blood pressure during the operation

Other medullary tumours include the *ganglioneuroma* which is benign and the *neuroblastoma* which is very malignant The latter affects children *When the right suprarenal is involved metastases occur*

systolic blood pressure much over 100 mm Hg renders the diagnosis of Addison s disease very unlikely unless the patient has previously suffered from hypertension

**Course and Complications** The course if untreated is usually progressive and complications such as intercurrent infections or myocardial degeneration may ensue Crises may occur in which the patient is collapsed and appears moribund Death may occur from hypoglycæmic coma

**Prognosis** This has been much improved by modern treatment The most favourable cases are those in which the suprarenal lesion is simple atrophy Active pulmonary tuberculosis is a very grave complication

**Treatment** Mild cases may be treated by the administration of salt alone given either as enteric coated 1 G tablets up to 5 tablets t i d or in milk or water flavoured with syrup of lemon In practically all cases however hormone therapy is indicated The best treatment appears to be the oral administration of cortisone tablets mg 12 5 to 25 daily in divided doses at 8 a m and 3 p m combined with the implantation of four mg 75 pellets of DOCA Substances rich in potassium should be avoided they include peas beans lentils soups dried fruits and chocolate

Should a crisis occur the patient should be kept warm and the bed heated with an electric cradle Cortisone or hydrocortisone mg 100 should be injected intravenously and as soon as the patient can swallow he should be given cortisone by mouth in doses of mg 100 to 200 in 24 hours Alternatively an intravenous drip should be given using a solution containing dextrose 100 G sod chlorid 10 G sod citrat 5 G Eucortone 50 ml and water to 1 000 ml Up to 150 ml Eucortone may be required in the 24 hours

Operation may sometimes be required for the removal of a tuber culous suprarenal gland and kidney The patient should be stabilised as described above and on the morning of the operation an intravenous injection should be given of 2 litres of the above solution with Eucortone 20 ml per litre After the operation this should be repeated once or twice

## THE PITUITARY BODY

**Introductory** The pituitary body is essential to life It is com posed of the following parts *The anterior lobe* (pars distalis) formed of chromophobe or agranular and chromophil or granular cells in columns the latter being eosinophil or α cells and basophil or β cells with con nective tissue and blood vessels In cases of adrenal cortical deficiency there is a reduction in the number of basophil cells It is thought that the chromophobe cells are the mother cells of the granular cells from which a secretion is derived The anterior lobe produces many hor mones the most important of which are the growth hormone the gonadotropic the thyrotropic the adrenocorticotropic (ACTH) the parathyrotropic and the lactogenic or luteotropic hormones Pancre atotropic diabetogenic and ketogenic hormones were formerly described It is now thought that the growth hormone and the adrenocorticotropic

somewhat frequent and the temperature usually subnormal apart from active tuberculosis Hypoglycæmic episodes are not infrequent Calcification of the suprarenals may be seen in some cases on X ray examination of the abdomen The blood Microcytic hypochromic anæmia may be present or the blood may be viscid There may be a relative lymphocytosis with an eosinophilia up to 6% The blood urea and non protein nitrogen rise in the penultimate stage due to reduction in blood volume, hæmoconcentration and diminished renal blood flow, and so extra renal azotæmia ensues There is usually a fall in the sodium potassium ratio below the normal of 30 The Na–K ratio is $\frac{\text{mEq Na/L}}{\text{mEq K/L}} = \frac{150}{5} = 30$ The 17 ketosteroids in the urine are reduced in women and in men The basal metabolic rate is low The electro encephalogram often shows abnormally slow waves The following tests of adrenal cortical function may be carried out

1 Thorn s test The eosinophil response to ACTH Normally after the intramuscular injection of mg 25 ACTH the adrenal cortex increases the output of 11 17 oxysteroids which causes a marked fall in eosinophils of over 50% In adrenal cortical insufficiency the fall in eosinophils is less than 50% The circulating eosinophils normally number 100 to 300 per c mm The first count is made in the morning after a night s fast The ACTH is then injected breakfast is then taken and the second count is made 4 hours after the first

2 The 17 ketosteroid excretion response to ACTH This test takes 48 hours Normally the 17 ketosteroids excretion rises as the circulating eosinophils fall after the injection of ACTH In primary adrenal deficiency there is no increased excretion In adrenal deficiency secondary to anterior pituitary lesions there is a slight rise in the 17 ketosteroid excretion on the second day

3 The Robinson Kepler Power test This is not dangerous It consists of two parts the first concerned with the delayed excretion of water in Addison s disease the second with an increased excretion of chloride in the urine and relative retention of urea in the blood (See *Proc Staff Meetings Mayo Clinic*, 1941 16 577)

4 Twenty four hours starvation during which only water is taken by mouth will cause a fall in blood sugar of over 20 mg /100 ml and hypoglycæmic symptoms may develop

**Differential Diagnosis** The weakness and pigmentation may suggest pernicious anæmia , this is excluded by a blood count Other causes of pigmentation and gastric disturbance must be considered such as arsenic poisoning abdominal growths and pregnancy Anorexia nausea and vomiting may suggest chronic cholecystitis or appendicitis Carcinoma of the stomach is excluded by the fractional test meal opaque meal occult blood test in fæces, and the absence of a tumour or of secondary deposits It may however co exist with Addison s disease In suspected arsenical poisoning the hair should be tested for arsenic Other causes of pigmentation which may have to be considered are race, sunburn, dirt, silver and lead poisoning hæmochromatosis abdominal tuberculosis, acanthosis nigricans and Graves' disease A

*The Pars Tuberalis* is difficult to demonstrate in man its function is not known

*The Pars Intermedia* This is very variable in amount It surrounds the pars nervosa and contains basophil cells It secretes intermedin which has a melanophore expanding effect on frogs and may also possess antidiuretic properties

*The Pars Nervosa* consists of neuroglial cells ependymal cells and pyramidal granular cells called pituicytes There are also present non myelinated nerve fibres hyaline masses and blood vessels Pituitrin is secreted probably from the pituicytes This has been subdivided into oxytocin (Pitocin) stimulating the uterus and vasopressin (Pitressin) which is antidiuretic, raises the blood pressure stimulates intestinal and other involuntary muscle and possibly raises the blood sugar by its effect on the glycogen in the liver and is concerned with fat meta bolism in the liver preventing obesity The secretion enters the blood stream and the posterior lobe secretions are not found in the cerebro spinal fluid

The anterior lobe pars tuberalis and pars intermedia constitute the glandular division and are derived from Rathke s pouch of the stomodæum and the pars nervosa infundibular stem and median eminence constitute the neural division and are derived from the floor of the fourth ventricle The posterior lobe is composed of the pars intermedia and the pars nervosa Disturbance of function is known as dyspitui tarism which may be in the sense of overactivity of any part or parts of the gland constituting hyperpituitarism or underactivity known as hypopituitarism

## Hyperpituitarism

Overactivity of the pituitary may give rise to sexual precocity or gigantism if it occurs before growth has ceased or to acromegaly if it begins later

In infancy hyperpituitarism may cause hemihypertrophy of the whole of one side of the child or one leg only may be affected This usually becomes unnoticeable as the child grows

During childhood gigantism (height over 79 inches) may ensue or pituitary glycosuria Thus a boy of 9 years of age may be over 6 feet tall

In adult life acromegaly may develop or virilism in women asso ciated with a basophil adenoma of the anterior lobe *Cushing s disease* is associated with such a tumour which may be very minute Young people are chiefly affected Males are usually tall and females short There is adiposity of the face neck and trunk but not of the limbs Purplish cutaneous striæ are seen on the lower part of the abdomen and on the thighs The blood pressure is raised and there is hypertrichosis in women of the chin upper lip and side whisker areas The pubic hair has a male distribution Hair tends to fall out on the head Erythrocytosis occurs in some cases The bones may be soft In some cases there is glycosuria There is usually impotence or amenorrhœa In addition to the pituitary lesion the testes or ovaries

*hormones* influence carbohydrate metabolism and that the growth hormone also exerts a ketogenic effect When the growth hormone is injected into dogs it causes diabetes The islets of Langerhans are thought to atrophy as the result of increased output of insulin to deal with the carbohydrate which is not utilised by the tissues

The growth hormone is thought to be produced by the eosinophil cells *ACTH* stimulates all the adrenal cortical hormones except aldosterone It is much more powerful when given intravenously than when injected intramuscularly The gonadotropic hormones are believed to influence both the female and male genital systems One hormone *Prolan A* known also as the follicle stimulating hormone (F S H ) in the *female* causes growth of the ovarian follicles and the liberation of the œstrogenic hormone œstradiol In the male it acts on the testis and is concerned with spermatogenesis The other hormone Prolan B or the luteinizing hormone (L H ) is responsible for the formation of the corpus luteum and the secretion of the ovarian hormone progesterone In the male it is concerned with the descent of the testicle and provokes the internal secretion of the interstitial cells which is known as testosterone Œstradiol is excreted in the urine both of women and men, and in women the amount excreted increases during pregnancy and during certain phases of the menstrual cycle Progesterone is excreted in the urine as pregnanediol, and testosterone is excreted as androsterone The presence in the urine of an anterior pituitary like luteinizing gonadotropic substance originating in the chorionic tissue of the placenta forms the basis of the Aschheim Zondek test for pregnancy It produces hæmorrhagic follicles in the ovary of the sexually im mature mouse A similar substance is found in the urine in chorion epithelioma, hydatidiform mole, and malignant disease of the testis *Œstrogens* are a group of complex substances chemically related to sterols They are excreted into the blood stream by the ovaries pla centa and suprarenal cortex during the reproductive period of the female The secretion is under the control of the anterior pituitary The œstro gens are responsible for the development of the secondary sex charac teristics They are necessary for pregnancy, parturition and lactation *Androgens* are similar chemically to œstrogens and are secreted by the testes and suprarenal cortex They are responsible for the male secon dary sex characteristics

*Clinical Applications of the Gonadotropic Hormones* The available preparations are not usually derived from the pituitary but from the serum of pregnant mares and from pregnancy urine They are pre sumably formed in the placenta Those obtained from pregnant mares' serum more completely resemble the two pituitary gonadotropic hormones than do those derived from human pregnancy urine, which are chiefly luteinizing in character The former include preparations such as Antostab, Gestyl Gonandyl and Serogan and those from pregnancy urine include Antuitrin S Gonan Physostab Pregnyl, and Prolan They are used for the treatment of pituitary infantilism, un descended testis and sexual under development, and for certain types of metrorrhagia and sterility



may be found atrophied post mortem The adrenals are often enlarged and may contain secondary adenomata Cushing's syndrome when occurring in women, is difficult to diagnose from the adreno genital syndrome The special tests described on p 697 should be carried out namely estimation of androgen excretion in the urine, and X ray examination of the kidneys before and after Pyelectan injection The 17 ketosteroids excreted in the urine are usually diminished in Cushing s disease due to a pituitary adenoma In the adrenogenital syndrome there are higher levels of urinary 17 ketosteroids Arrhenoblastoma of the ovary is another cause of hirsuties, but in this rare condition the patient is not obese, cutaneous striæ if present, are not purplish and the blood pressure is seldom raised X ray treatment to the skull may afford relief A pituitary tumour (usually an agranular adenoma) may cause pressure symptoms such as loss of vision and headache without symptoms of secretory disturbance If it presses on the basophilic tissue sexual deficiency symptoms such as amenorrhœa may occur There may be optic atrophy with leg pains and absent knee jerks (pituitary tabes) differing from tabes dorsalis clinically in that the pupils react to light

Stalk tumours (suprasellar cysts) cause headache and loss of vision The sella turcica is often distorted and a shadow may be seen above it by X rays There may be early optic neuritis and later atrophy clinically it closely resembles an adenoma of the pituitary A suprasellar endothelioma may also occur

*Pituitary Glycosuria* The mechanism of its production has been described on p 702 Pituitrin also inhibits the action of insulin Clinically pituitary glycosuria may be associated with a pituitary tumour or with basal meningitis

## Acromegaly

**Definition** A disease characterised by enlargement of the extremities

**Etiology** Acromegaly is caused by oversecretion of the pituitary (chiefly anterior lobe)

**Pathology** There is usually an eosinophil adenoma of the anterior lobe less frequently the tumour is a glioma endothelioma or sarcoma There is often hyperplasia of the adrenal cortex

**Clinical Findings** The patient is usually between the ages of 20 and 40 at the onset The disease may first show itself directly after pregnancy The sex incidence is practically equal The patient may first notice tingling or numbness of the hands and feet and later enlarge ment of the head face hands or feet He then complains of headache often bi temporal and of visual disturbances such as dimness of the outer part of the fields of vision Amenorrhœa may be the first symptom Early there may be sexual excitement but mental torpor develops later There may also be loss of smell, trigeminal neuralgia or nasal discharges of blood, mucus or cerebro spinal fluid In some cases the patient has attacks known as uncinate fits " with an aura of smell or taste and convulsions may follow

*On Examination* In an established case the appearance is characteristic the lower jaw the malar bones and orbital ridges are prominent and the skull is enlarged Twenty per cent. of patients are over 5 feet 11 inches in height The tongue is big the teeth spaced the nose broad the skin rough thick and it may be dark The hands and feet are enlarged the chest big and there is frequently kyphosis Pierre Marie described two types of hand the *type en long* and the *type massive* which is spade like (see Fig 67) The thyroid gland is often palpable The temperature is low in the later stages of the disease The blood pressure may be raised

*Special Examinations* The eyes The characteristic change is bi temporal hemianopia due to pressure of the tumour on the decussating fibres of the optic nerve in the chiasma This is determined by plotting the fields of vision Early changes include primary optic atrophy and a scotoma for red Optic neuritis rarely results from generalised increased intracranial pressure The third fourth or sixth cranial nerves may be compressed in the cavernous sinus with weakness of the ocular muscles A stereoscopic X ray of the skull This may show enlargement of the sella turcica or erosion of one or both of the anterior or posterior clinoid processes X ray of the hands A tufting appearance may be seen in the terminal phalanges which show lines radiating like a fan Sugar tolerance test In the early stages the tolerance may be diminished the blood sugar rising above the renal threshold and glycosuria resulting Later there is hypopituitarism with increased sugar tolerance Basal metabolic rate This may be increased early and diminished later in the disease

Differential Diagnosis The appearances of a developed case are typical In the early stages the special examinations mentioned above will help in the detection of a pituitary lesion

Course and Complications The course is usually progressive but there may be intermissions Certain types are described 1 *Benign* Duration up to 50 years 2 *Chronic* Duration 8 to 30 years 3 *Acute malignant* Duration 3 to 4 years 4 *Cephalic* Head only affected 5 *Stationary* 6 *Formes frustes* The headaches may become excruciating and blindness or dementia occur Death may result from heart failure convulsions diabetes mellitus uræmia or intercurrent infections such as pneumonia and tuberculosis

Prognosis This is very grave but there is a tendency to arrest in some cases and a cure has been effected by operation

Treatment X ray treatment to the skull may afford relief The use of stilbœstrol mg 5 or more daily or of testosterone mg 50 daily to lower the eosinophil content of the pituitary is under investigation Operation is usually advised on account of threatened loss of vision or severe headache A decompression and incision of the capsule of dura may relieve headache even if the tumour is not removable Insulin and dietetic restrictions may be necessary for the glycosuria In the later stages of hypopituitarism daily subcutaneous injections may be given of 1 ml of anterior lobe extract (Antuitrin)



Fig 6" Acromegaly

with trophic changes in the teeth hair and nails loss of appetite vomiting and profound weakness The axillary and pubic hair are frequently lost Sexual power disappears, the temperature and B M R are low The similarity to anorexia nervosa has been referred to In some respects it resembles Addison s disease but there is usually no pigmentation In others it manifests itself as pituitary myxœdema or pituitary hypothyroidism If untreated death usually occurs in 1 to 2 years in some cases due to hypoglycæmic coma All grades of Simmonds disease exist from the very mild to the very severe Complete recovery may occur if the patient becomes pregnant again In panhypopituitarism the insulin sensitivity test shows hypoglycæmia unresponsiveness and the 17 ketosteroid excretion is usually low Hypoglycæmia unresponsiveness also occurs in Addison s disease

*Dercum s Disease* (Adiposis dolorosa) This usually occurs in women after the climacteric Painful fatty subcutaneous masses appear especially in the upper arms and thighs and there is general weakness and mental deterioration In all cases of suspected hypopituitarism a sugar tolerance test should be performed The tolerance is usually increased The basal metabolic rate is low An X ray film of the skull may reveal alterations in the pituitary fossa and visual disturbances may occur

**Treatment** This is not very satisfactory If a tumour is present an operation may be necessary to relieve headache or preserve vision if the tumour is insensitive to irradiation In pituitary infantilism treatment should be begun with gr ½ of thyroideum Oral administration of pituitary extract is probably without effect Children may be given subcutaneous injections of Antuitrin Growth 2 to 5 ml twice a week A gonadotropic factor obtained from pregnant mares serum such as Antostab can also be injected 1 ml (200 i u ) daily In Simmonds disease cortisone should be given by mouth in doses of mg 12 5 one to three times daily Thyroideum gr ¼ gradually increased to gr 1 or gr 1½ daily, should be given after 10 days treatment with cortisone

## Diabetes Insipidus

**Definition** A disease characterised by thirst and persistent polyuria the urine being of low specific gravity and containing no abnormal substance

**Etiology** Probably all cases are due to lack of the anti diuretic pituitary hormone associated with hypofunction of the posterior lobe or with lesions of the hypothalamus near the tuber cinerium The nucleus supraopticus in the hypothalamus is probably the controlling centre Water balance in the body is disturbed by loss of the oliguric hormone which normally affects the thin segment of the loop of Henle stimulating it to absorb water and so to concentrate the urine Normally 168 5 litres of glomerular filtrate are reabsorbed in 24 hours and in diabetes insipidus this amount may be reduced to 130 litres The secretion of the thyroid gland also appears to increase water elimination and symptoms of diabetes insipidus are sometimes associated with thyro

## Hypopituitarism

The effects produced by under activity of the pituitary vary with the age of the patient The characteristic features are sexual inactivity infantilism, deposition of fat, lowered metabolism and oversecretion of urine The lesion, if an adenoma, is of the chromophobe type, and probably produces its effect by pressure on the chromophil cells of the pituitary In other cases the symptoms are due to a persistence of chromophobe cells and improvement occurs as more granular cells are formed

## Pituitary Infantilism

*The Lorain Type* (Ateliosis) The child does not grow the trunk especially being short The intelligence is good The sexual organs are small but there is no adiposity It is considered to be due to insufficiency of the anterior lobe

*Frohlich's Type* (Dystrophia adiposo genitalis) This was originally described in a boy of 14 and at operation a cyst the size of a hazel nut was found which was thought to be in the pituitary Histological examination suggested an adenoma of the pituitary There was destruction of the sphenoid and dorsum sellæ severe frontal headaches and blindness of the left eye The disease may not commence until adult life There is adiposity affecting the arms legs and trunk especially around the pelvis and below the scapulæ The adiposity is probably due to a lesion of the neighbouring hypothalamus The skin is smooth the fingers tapering the genital organs immature the intelli *gence is usually normal but there is a tendency to somnolence* (fat boy of Dickens) The blood pressure is often slightly raised The temperature is subnormal and the sugar tolerance is increased Males tend to conform to the female type This variety occurring in child ren is sometimes called *the Brissaud type* Another variation occurring in children in which the face is round is called the pudding face type of Fearnsides They are thought to be due to insufficiency of both lobes of the pituitary *The Laurence Moon Biedl syndrome* is characterised by obesity polydactylysm mental deficiency and retinal changes resembling those of retinitis pigmentosa

## Hypopituitarism in Adults

There is usually general obesity especially around the pelvic girdle and diminution of sexual function The male skeleton and pubic hair distribution tend to conform to the female type The fingers become delicate and tapering and the skin smooth In females there is usually amenorrhœa

*Simmonds Disease* (Panhypopituitarism, anterior) This is due to deficiency of the anterior lobe which is often found to be replaced by scar tissue or which may be the site of an embolus thrombosis or *hæmorrhage* The original case was associated with puerperal sepsis It may also follow a severe post partum hæmorrhage or be caused by tumours or cysts It occurs in adults usually over the age of 30, and affects both sexes There is usually but not always marked emaciation

at night or 0 5 ml of Pitressin which is the active constituent If intestinal colic ensues 1 ml of Pituitrin (half strength) may be given, as a nasal spray, or on a little wool as a nasal plug as an alternative, but this is not always efficacious Nasal insufflation of the powdered whole gland, mg 25 3 or 4 times daily and during the night may also be tried A more prolonged effect lasting for about 44 hours is obtained by the subcutaneous injection of 1 to 1 5 ml of Pitressin Tannate (5 pressor units in 1 ml of peanut oil) This treatment may have to be continued indefinitely Thyroidectomy has proved successful in certain cases following encephalitis lethargica and in those associated with thyrotoxicosis

## THE THYMUS GLAND

The thymus increases normally in size until puberty after which it gradually atrophies The thymus may hypertrophy if the adrenals atrophy

### Status Lymphaticus

A condition characterised by enlargement of the thymus gland and lymphatic tissue generally The symptoms include Thymic asthma, due to pressure of the enlarged gland causing dyspnœa with stridor This may be mistaken for croup due to laryngeal obstruction Sudden death may occur during sleep or during the administration of an anæsthetic possibly due to mechanical stimulation of the vagus A status hypoplasticus It is thought that some children with the status lymphaticus grow up there being a compensatory overactivity of other glands such as the suprarenal or pituitary (Timme s multi glandular syndrome) Enlargement of the thymus can be diagnosed by X ray examination and treated by exposure to X rays

### Thymic Tumours

These may be simple such as a lipoma or fibroma cystic such as a dermoid or malignant such as a carcinoma or sarcoma The tumour may also be due to Hodgkin s disease The effects produced are mech anical those of a superior mediastinal tumour In myasthenia gravis small non malignant soft encapsulated nodules are found in some cases A thymic tumour may be shown by lateral tomography

Treatment The tumour may be removed in some cases by an operation involving splitting of the sternum If this is not possible X rays should be used

## THE PINEAL BODY

The function of the pineal body is not definitely known It probably forms no internal secretion but exerts through the nervous system a check upon sex development

### The Pineal Syndrome

A pineal tumour such as a teratoma may give rise to overgrowth and sexual precocity in boys but not in girls It may also produce

toxicosis *Predisposing causes* 1 Heredity especially in the benign idiopathic cases 2 Age 10 to 40 years 3 Sex Males predominate

**Pathology** The following lesions have occurred Sarcoma secondary carcinoma a chromophobe adenoma sarcoid sphenoidal sinusitis gumma or leukæmic infiltration of the pituitary injury to the sella turcica as by a bayonet or bullet wound or fractured base basal syphilitic or tuberculous meningitis involving the inter peduncular space It is also a symptom of the Hand Schuller Christian disease and of post encephalitic Parkinsonism Experimental puncture of the hypo thalamus or compression of the infundibulum by a clip results in diabetes insipidus At autopsy there may be enlargement of the kidneys ureters and bladder

**Clinical Findings** The patient is usually a young adult, who notices increasing thirst and the passage of large quantities of urine There may be a sudden onset after an injury He is usually constipated the mouth is dry and sleep is disturbed by the desire for micturition Sometimes there is great hunger and thirst becomes unquenchable

*On Examination* In an established case there is usually emaciation with a dry skin Neighbourhood symptoms may be present if the lesion is a tumour The urine Specific gravity 1 001 to 1 005 Volume 10 to 40 litres daily There is no albumin but occasionally a trace of sugar may be present The blood The red cells may be over 6 millions per c mm The Wassermann reaction may be positive The sugar tolerance is normal or diminished The basal metabolic rate is usually normal An X ray film of the skull may show enlargement of the sella turcica

**Differential Diagnosis** Other varieties of polyuria and frequency must be excluded especially those due to chronic nephrosclerosis inter mittent hydronephrosis, diabetes mellitus hysteria and an enlarged prostate The renal function tests and abnormal urinary constituents serve to diagnose chronic nephritis In hysteria and intermittent hydronephrosis the polyuria is inconstant and a renal swelling may be felt in the latter Examinations of the urine and the blood for sugar differentiate diabetes mellitus The nicotine test may be used as a diagnostic procedure Normally the inhalation of the smoke of 1 to 3 cigarettes until there is severe malaise giddiness nausea or vomiting causes an antidiuretic response owing to stimulation of the supraoptico hypophyseal system and release of the antidiuretic hormone In many cases of diabetes insipidus no such antidiuretic response is obtained

**Course and Complications** The hereditary type often runs a prolonged course for many years In other cases the course must vary with the cause Complications include pulmonary tuberculosis and coma the latter is the usual mode of termination

**Prognosis** This varies with the cause In idiopathic cases life may be little, if at all curtailed Syphilitic cases may be cured by treat ment In malignant cases the disease is usually fatal within a year

**Treatment** In syphilitic cases iodides and penicillin should be given A salt poor diet may be given but the fluid intake should not be restricted Pituitrin has an antidiuretic effect 1 ml may be required twice or three times a day intramuscularly or last thing

at night or 0 5 ml of Pitressin which is the active constituent If intestinal colic ensues 1 ml of Pituitrin (half strength) may be given, as a nasal spray, or on a little wool as a nasal plug as an alternative, but this is not always efficacious Nasal insufflation of the powdered whole gland mg 25 3 or 4 times daily and during the night may also be tried A more prolonged effect lasting for about 44 hours is obtained by the subcutaneous injection of 1 to 1 5 ml of Pitressin Tannate (5 pressor units in 1 ml of peanut oil) This treatment may have to be continued indefinitely Thyroidectomy has proved successful in certain cases following encephalitis lethargica and in those associated with thyrotoxicosis

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The function of the pineal body is not definitely known It probably forms no internal secretion but exerts through the nervous system a check upon sex development

### The Pineal Syndrome

A pineal tumour such as a teratoma may give rise to overgrowth and sexual precocity in boys but not in girls It may also produce

internal hydrocephalus with headache owing to obstruction of the Sylvian aqueduct, pressure on the corpora quadrigemina or cerebellum may result in diplopia and fits, and may cause tremors of the extremities One or other pupil may not react to light and external *oculo* motor paralysis may be present X rays may reveal a pineal shadow, but the pineal body is calcified in about 60% of normal people over 25 years of age The prognosis is usually hopeless as surgical removal of the tumour is rarely possible X ray treatment may be given

## THE TESTES

As described above the luteinizing hormone of the anterior pituitary is concerned with the descent of the testicle and provokes the internal secretion of the interstitial cells which is known as testosterone Testosterone can be synthesised and its propionate is available for clinical use The treatment for failure of descent of the testicles where there is no mechanical obstruction consists in the injection of a preparation obtained from pregnancy urine such as Antuitrin S, 1 000 to 1 500 i u twice a week for 3 months Testicular atrophy may be primary or secondary to hypopituitarism, injury or to such infections as mumps In both types there may be hypogonadism due to lack of androgens deficient spermatogenesis or hypogonadism combined with hypospermia Sperm counts and testicular biopsies serve to differentiate Hypogonadism due to primary atrophy is treated with testosterone propionate mg 5 to 50 being injected intramuscularly daily Methyl testosterone may be given by mouth daily mg 10 to 100 being usually required A subcutaneous implant of mg 300 of testosterone will be effective for about 2 months Atrophy secondary to hypopituitarism is treated by chorionic gonadotropin Antuitrin S 1 000 to 1 500 international units (500 i u /ml ) may be injected twice a week for 3 months Testosterone must not be given in these cases as it inhibits the pituitary production of gonadotropins

## THE OVARIES

*Ovarian Hormones* As described above the ovary, when stimulated by the pituitary gonadotropic hormones secretes the œstrogenic hormone œstradiol and the active principle of the corpus luteum progesterone Œstradiol is concerned with the growth of breast duct tissue and with the proliferative changes in the uterine wall which occur during the first half of the menstrual cycle Progesterone causes premenstrual changes in the uterine mucosa is responsible for the implantation of the ovum and placental formation and growth of the alveolar breast tissue during pregnancy Œstradiol which is regarded as the primary hormone is not detectable in the urine Instead the structurally related but physiologically less active compounds œstrone and œstriol are found Urinary extracts do not contain measurable amounts of progestational substances Progesterone cannot be isolated from late pregnancy urine or blood as such but the biologically inactive reduction products pregnonediol can be obtained

Synthetic oestrogenic analogues (such as stilboestrol, hexoestrol and dienoestrol) are also now available for clinical use Progesterone is obtained from the corpora lutea of sows or it can be synthesised

*Clinical Applications* The value of these oestrogenic substances in the treatment of amenorrhoea is doubtful but primary amenorrhoea with infantilism has been treated by the injection of 250 000 international benzoate units of oestradiol spread over a period of 5 weeks To inhibit lactation after childbirth stilboestrol mg 5 to 10 given by mouth within 24 hours of the birth is usually sufficient Mg 1 to 5 t d s are required for the treatment of kraurosis and senile vaginitis and for vulvo vaginitis in infants and children mg 1 tablet crushed in milk is given t i d for 12 doses Symptoms due to deficiency of ovarian secretion occur at the climacteric They include amenorrhoea headache flushings increase of weight and nervous instability They usually pass off in the course of 2 or 3 years The nervous symptoms can be relieved to a certain extent by the administration of bromides such as sodium bromide gr 5 to 10 t d s The administration by mouth of stilboestrol in doses varying from mg 0 1 to 5 t i d is very successful The dose should be reduced as soon as the symptoms improve Progesterone is used for the treatment of threatened abortion (mg 5 to 10 daily for a short period) for habitual abortion (mg 1 to 2 daily until the thirty second week of pregnancy) and it is sometimes of value in menorrhagia (mg 1 to 5 daily for 6 days) The symptoms of *premenstrual tension* can be relieved by the intramuscular injection of progesterone mg 10 or by mouth tab acthisterone mg 10 t d s every other day for 5 to 7 days before the period is due and by the administration by mouth of aethinyloestradiol mg 0 05 tab for 21 days after the cessation of each period

## INFANTILISM AND DWARFISM

Definition Infantilism is a condition characterised by delay of adult development with deficiency of secondary sexual characteristics There may be no abnormality of growth In dwarfism the secondary sexual characteristics mental and bodily are present but growth is stunted

### Infantilism

This may be due to 1 *Deficiency of an internal secretion* Thyroid deficiency as in cretinism pituitary deficiency as in the Lorain type or Frohlich s type of infantilism An infarct in the anterior lobe of the pituitary will also cause progeria Pancreatic deficiency as in pancreatic infantilism with inefficient fat digestion renal deficiency as in renal infantilism associated with cardiorenal degeneration and dilatation of the bladder ureters and pelvis of the kidneys testicular and ovarian deficiency as in male and female eunuchism 2 *Mongolism* This may occur especially in the last born child in a large family or if the mother is elderly There is mental deficiency and the appearance of the child is characteristic The palpebral opening slants downwards and inwards the hands are broad the index finger is short and the little finger

curved with concavity to the radial side In about 27% of cases two creases only instead of the normal three creases, are present on the little finger of one or both hands, the distal and medial creases being replaced by a crease between the distal and proximal inter phalangeal joints (Penrose) The foot "grasp reflex" is present in about 5% of cases The tongue is often large fissured and pro trudes The disposition is usually calm and affectionate Enlargement of the thyroid gland and congenital heart disease may be present The condition is incurable and not benefited by thyroid treatment 3 *Congenital aberrations and diseases* such as microcephaly, congenital heart disease, anangioplastic infantilism (the arteries being improperly developed) persistent thymus and congenital syphilis 4 *Acquired disease and infections* such as extensive bronchiectasis, ankylostomiasis malaria and alcoholism

## Dwarfism

This may be due to such conditions as rickets renal dwarfism spinal caries osteogenesis imperfecta achondroplasia, phocomelus (φωκη = a seal, μελος = limb) hydrocephalus and microcephaly

## CHAPTER XIII

## THE TROPICAL DISEASES

### Malaria

**Definition** A disease characterised by recurrent attacks of fever, due to infection with a specific protozoon

**Etiology** Malaria is due to infection with a protozoon the Plasmodium malariæ Infection in man is caused by the bite of a female anopheline mosquito The mosquitoes may fly normally 1 to 2 miles or as far as 10 miles if aided by the wind Four types of plasmodium affect man, the *P vivax* causing benign tertian malaria the *P malariæ* causing quartan malaria and the *P falciparum* causing malignant tertian malaria The *P ovale* is an uncommon parasite so called because the infected red cell becomes oval shaped It causes a tertian fever The incidence of malaria in a locality can be gauged by the spleen index *i e* the proportion of the population with a palpable spleen and the parasitic index as shown by blood slides These run very closely parallel

*The Life Cycles of the Parasite* The mosquito forms the definitive host and man the intermediate host There are two cycles an asexual cycle in man and a sexual cycle in a mosquito

*The Asexual Cycle* The mosquito when it bites man introduces *sporozoites* Within 2 hours the *sporozoites disappear from the blood* and settle in fixed tissue cells particularly in the liver They now pass through the schizont stage and merozoites are discharged into the blood This is known as the pre erythrocytic or PE phase of the parasite After about a week these enter the red cells and become trophozoites These grow and divide into segments forming schizonts (rosette) which rupture liberating merozoites into the blood The merozoites may now enter other red cells forming trophozoites thus completing the asexual cycle This constitutes the erythrocytic or E cycle of the parasite and causes the clinical attack of malaria The majority of the parasites are in the capillaries of the spleen and bone marrow and not in the peripheral circulation

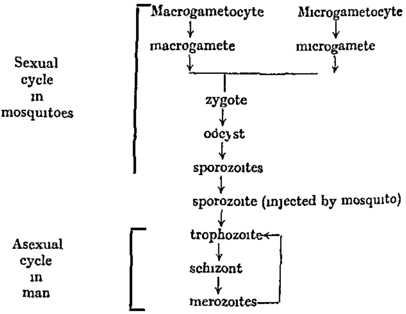
*The Sexual Cycle* Some of the trophozoites may enlarge and become macro or microgametocytes If these remain in the blood they die without further development If however a mosquito bites a man when the gametocytes are present they are taken into the mosquito s stomach where further development occurs The macrogametocyte becomes a female gamete and the microgametocyte forms a microgamete A microgamete enters and fertilises a macrogamete and a zygote results This passes through the mucous membrane of the mosquito s stomach and forms an oocyst Numerous sporozoites develop in this cyst which finally ruptures into the abdominal cavity and the sporozoites pass to the salivary glands of the mosquito and are injected into man by the

mosquito's bite In *P falciparum* infections the PE phase is thought to disappear soon after the E cycle is completed Relapses are due to E forms persisting in the blood In *P vivax*, *P malariæ* and *P ovale* infections the infecting agent remains in the liver as the exo erythrocytic or EE phase and relapses are probably due to merozoites liberated from this source

These stages can be represented diagrammatically as shown below

The main distinguishing features between the types of parasite are as follows

*Plasmodium Vivax* This causes benign tertian malaria The duration of the asexual cycle in man is 48 hours The schizont is like a




rosette with about 18 merozoites Schuffner s dots (deeply staining) are seen inside some of the red cells, which are enlarged and pale The gametocytes are large and round

*Plasmodium Malariæ* This causes quartan malaria The duration of the asexual cycle in man is 72 hours the schizont is like a daisy head, with 6 to 12 merozoites The red cells are normal The gametocytes are large and round

*Plasmodium Falciparum* This causes æstivo autumnal or malignant tertian or subtertian malaria The duration of the asexual cycle in man is probably 48 hours Schizonts are rarely seen in the peripheral blood, but they contain about 20 irregularly arranged merozoites The gametocytes are crescentic, the female ones having more pointed ends than the male *Predisposing causes* 1 Locality Malaria is rife in the tropics, especially in India Africa and America It also occurs in Southern Europe and is occasionally met with in South east England

2 Temperature A mean temperature of 60° F for at least 2 weeks is necessary for the development of the protozoon in the mosquito Stagnant water favours breeding of the mosquitoes 3 Season and rainfall The effect of these upon the incidence of malaria varies in different localities 4 Age Children are especially susceptible but all ages may be affected

Pathology The spleen becomes enlarged and very hard in chronic cases (ague cake) In acute cases it is soft and swollen The liver may be enlarged and dark red The brain may be dark slate-coloured Parasites and pigment may be found in the vessels in various organs such as the spleen kidneys and brain

Clinical Findings The symptoms in the benign tertian and quartan types of malaria apart from the time intervals of the fever are usually indistinguishable The patient complains of periodical attacks beginning generally about noon or later of malaise shivering headache and perhaps nausea He feels cold all over and may vomit After a varying period of about 2 hours the skin becomes hot and flushed and the headache is more intense This phase persists for 2 or 3 hours and is then followed by sweating and marked relief from the discomforts

*On Examination* During the initial cold stage the skin is blanched, but the body temperature rises to about 102° F or 104° F and the pulse is frequent The fever is thought to correspond with the liberation of the spores in the blood perhaps due to freeing of their toxins but there must be a threshold value a definite amount of toxin being required in any individual to produce a rise of temperature Thus patients may have schizonts in the peripheral blood without any fever and again there may be fever without schizonts being found although they are probably present deeper in the body In the hot stage the temperature begins to fall and in the sweating stage it reaches normal or subnormal The whole attack lasts about 12 hours

In the benign tertian infection the pyrexial attacks follow each other every third day in the quartan every fourth day (regularly inter mittent fever) Daily (quotidian) attacks may be due to a double infection with benign tertian or to a threefold infection with quartan malaria or in some cases to malignant infection A mixed infection may cause irregular attacks of fever

In malignant (subtertian) malaria attacks are liable to occur in the summer or autumn in temperate zones but in the tropics this seasonal incidence does not prevail In the less severe types the attacks may recur daily or every other day or at irregular intervals Frequently there are no rigors but the fever may last for 24 hours or longer Specially severe variations include (*a*) Cerebral malaria in which the patient rapidly becomes unconscious with hyperpyrexia (temperature over 107 F ) In other instances there may be convulsions paralysis or meningitic symptoms (*b*) Algid type The skin remains cold and the body temperature may be subnormal or raised above the normal There is great collapse and weakness In some cases there is vomiting or diarrhœa or hæmorrhages into the skin and various organs (*c*) A bilious remittent fever The characteristic features are the continuous

temperature, jaundice and biliary vomiting There is usually severe epigastric pain In the typhoid type the patient is more ill the tongue is dry and delirium present Blackwater fever is probably a variety of malignant malaria

The spleen is usually palpable during the paroxysms, and in persistent cases remains so between the attacks eventually becoming hard It may reach to the umbilicus The blood When the tempera ture rises a film should be made and stained for malarial parasites Examination between the attacks usually yields negative results In chronic cases a hæmolytic anæmia develops, and the large mononuclears may increase up to 20% A positive Wassermann reaction may be obtained after the attack but it is not universally accepted that this is due to the malaria An indirect van den Bergh reaction is present in a large proportion of uncomplicated cases of subtertian malaria The urine Albumin is present in a few cases and hæmoglobinuria is the characteristic feature of blackwater fever

**Differential Diagnosis** In a malarial country malaria is the cause of the majority of feverish attacks but when the patient has resided out of a malarial district for several years the infection dies out Malaria must be diagnosed from other conditions, such as kala azar tuberculosis amœbic hepatitis typhoid fever, sunstroke meningitis, cholera dysentery, and pyæmic infections causing rigors The diagnosis rests upon (a) Finding the parasite in the blood Prophylactic doses of quinine or mepacrine obscure the diagnosis as then no parasites are likely to be present in the peripheral blood (b) The response to anti malarial drugs If the temperature does not fall after their adequate administration the pyrexia is almost certainly not due to malaria (c) The presence of a leucocytosis is also evidence against malaria (d) Herpes labialis is frequently noted in an attack of malaria

**Course and Complications** The course depends largely upon the treatment the attacks in the benign tertian and quartan varieties are usually readily controlled but there is more difficulty with the malig nant type In certain localities blackwater fever is liable to ensue in patients suffering from chronic malaria or in those who have been improperly treated Relapses, where fresh infection is impossible may occur at considerable intervals after the last infection and may be due to the schizonts lurking in the internal organs or being present in too small numbers in the peripheral blood to be detected Malarial cachexia is a troublesome feature of repeated attacks Complica tions include Bronchitis labial herpes neuralgia neuritis asthma glycosuria and pneumonia Pulmonary tuberculosis may develop as a sequela Blackwater fever is probably a complication of malignant malaria

**Prognosis** A favourable prognosis depends largely on early diagnosis and adequate treatment The outlook is usually very grave in the cerebral and algid types of malignant malaria Death is most often due to cerebral malaria

**Treatment.** *Prophylactic* This is concerned with (a) Elimination of the breeding grounds of the mosquitoes This is best effected by

drainage and by sprinkling stagnant pools with some substance to destroy the larvæ of the mosquitoes D D T powder sprayed from aeroplanes is effective for 1/10 lb per acre on water will destroy all mosquito larvæ (*b*) Destruction of mosquitoes in houses or cattle sheds during the winter (*c*) Prevention of mosquito bites by the use of electric fans sleeping nets and protection of the ankles during the evening or early morning by two pairs of socks or Wellington boots Dimethyl phthalate may be applied as an insect repellent in the form of a liquid *cream or lotion to the skin or clothing It should not be used* on artificial silk garments (*d*) The prevention of infection in non immunes *i e* those who go to an endemic area and who have not previously suffered from malaria is a matter of some importance The drugs most often used are chloroquine diphosphate (Aralen) mepacrine (Atebrin) proguanil (Paludrine) and quinine only if these drugs are not available

Chloroquine is given in doses of 2 tabs (= 300 mg base) once a week for an adult mepacrine or proguanil 1 tablet (mg 100) daily Such prophylactic treatment should be begun 2 weeks before entering a malarial country and continued for 4 weeks after leaving the endemic area

*Causal prophylaxis, i e* destruction of the parasite before it enters the red cell can probably only be effected in *P falciparum* infections This is achieved by the use of proguanil or of pyrimethamine (Daraprim) 1 tab mg 25 weekly In the other varieties of infection prophylaxis is effected by suppression of the parasite in the blood Relapses are therefore liable to occur for a time after cessation of treatment

*Curative* The patient should go to bed and be kept warm with blankets and hot bottles during the cold stage The treatment varies with the type of infection present For *P falciparum* infections chloroquine amodiaquine or mepacrine may be used In a severe case *chloroquine mg 200 in 5 ml ampoule is given intravenously* The contents are injected in 10 ml pyrogen free sterile distilled water very slowly intravenously The dose can be repeated after 8 hours up to 3 doses in 24 hours It is not likely to produce blackwater fever In less severe cases it is given by mouth in the following dosage 4 tabs (= 600 mg base) followed 6 hours later by 2 tabs On the next 3 days 2 tabs The dose of mepacrine is 3 tabs (= 300 mg ) t i d on the first day 2 tabs t i d on the second day and 1 tab t i d for the next 5 days Alternatively in a very severe case an intravenous injection may be *given very slowly of quinin dihydrochlorid gr 10 (0 66 G ) dissolved* in 10 to 15 ml of sterile pyrogen free water

The treatment of an acute attack of *P vivax P malariæ* or *P ovale* infections consists in the eradication of the E (erythrocytic) forms of the parasites by chloroquine or mepacrine as for *P falciparum* infections followed by attempts to get rid of the FE (exo erythrocytic) forms by the use of one of the 8 amino quinolines such as primaquin which is said to be less toxic than pamaquin The tablet of primaquin contains mg 7 or mg 15 of the base and is given for 14 days either mg 7 t i d or mg 15 once daily

## Blackwater Fever

### (*Malarial Hæmoglobinuria*)

**Definition** A complication of malaria, characterised by hæmoglobinuria

**Etiology** Blackwater fever is usually considered to result from chronic infection with subtertian malaria A chill, fatigue, or a dose of quinine, may precipitate an attack The destruction of the red cells is presumably due to a lysin, but no such lysin has been demonstrated in the peripheral blood Parasites are found in the blood in over 70% of cases before the onset of the hæmoglobinuria usually of the subtertian type When severe hæmolysis is taking place the parasites are difficult to demonstrate in the blood *Predisposing causes* 1 Locality Districts where the incidence of subtertian malaria is high and the disease is endemic It is thus especially prevalent in tropical Africa and in certain parts of India etc It occurs in England in patients who have returned after contracting subtertian malaria abroad 2 Nationality Europeans are more prone than natives 3 Repeated attacks of malaria The patient has usually lived over 6 months in the tropical country 4 A previous attack of blackwater fever

**Pathology** The kidneys are enlarged and very congested The renal tubules are obstructed with *débris* The spleen and liver are enlarged soft and pigmented The brain and bone marrow may be pigmented and the heart show fatty changes The hæmolysis is believed to occur in the general circulation and not in the kidneys

**Clinical Findings** The patient is usually an adult European, who has been in a malarial country for over 6 months and who gives a history of repeated attacks of malaria which have not been treated efficiently The onset is generally sudden, with a rigor, the urine is then noticed to be red and there may be some frequency Feverish symptoms such as malaise thirst headache and loss of appetite are complained of There may be severe pain in the epigastrium or loins with nausea or vomiting of bile stained fluid Hiccough is a symptom in very severe cases

*On Examination* The patient is often jaundiced and the temperature is raised to 102 F or over The spleen is palpable and tenderness may be elicited over it in the epigastrium or renal areas The urine In some cases there is polyuria in others oliguria or anuria The urine varies in colour from almost black to pale red A dark deposit settles on standing The specific gravity is raised and the reaction is acid Albumin is present in considerable quantities The red colour is due to blood Met Hb being present and Oxy Hb in the severe cases The deposit consists of blood and granular casts epithelial cells and a few red cells The blood There is a hæmolytic anæmia of varying degree The red cells may be diminished by 50% within 24 hours and may show ghost forms and punctate basophilia The fragility of the red cells is normal The white cells The large mononuclears may show an increase up to 10% or more The indirect van den Bergh reaction is positive and in severe cases the direct reaction is also positive The blood urea

is raised Oxyhæmoglobin and methæmalbumin are present in the plasma

**Differential Diagnosis** The jaundice, temperature and bilious vomiting with dark urine are suggestive of yellow fever In yellow fever there is hæmatemesis and the urine contains bile Other causes of hæmoglobinuria rarely require elimination There is usually no difficulty in diagnosis

**Course and Complications** The hæmoglobinuria persists for a few hours or days and relapses are not infrequent The temperature falls to normal when the urine clears Complications include Suppression of urine and hyperpyrexia Cholelithiasis may ensue as a sequela

**Prognosis** There are many mild cases which recover but the outlook is very grave in any severe case

**Treatment** *Prophylactic* In districts where blackwater fever is endemic every attack of malignant tertian malaria should be adequately treated with chloroquine in doses given on p 717 or amodiaquine (Camoquin) in doses of tab mg 200 base first 4 tabs after 6 hours 2 tabs and then 2 tabs daily for 2 days

*Curative* The disease should never be treated lightly The patient must be put to bed and kept lying down at absolute rest He should be given fluids containing dextrose the amount of urine passed must be measured and charted daily and when diuresis occurs the volume of ingested fluid can be increased The intake of fluid should not exceed the output from the various sources such as vomit sweating urine and fæces except in very hot countries An alkaline mixture containing Sod bicarb gr 80 sod citrat gr 20 sp chlorof m 7 aq menth pip dest ad fl oz 1 should be given six hourly or four hourly The value of endeavouring to keep the urine alkaline has been disputed In some cases despite large doses the urine remains acid It is not now believed that oliguria or anuria is due to blocking of the renal tubules with precipitated hæmoglobin derivatives from an acid urine It is considered to be due to glomerular failure and renal anoxia If vomiting prevents this drip rectal salines containing sod bicarb gr 120 to 1 pint, should be given If there is oliguria hot applications such as water bottles should be applied to the loins An intravenous injection of 1 pint of 5% dextrose in normal saline or of 2% sodium bicarbonate in normal saline may prove helpful If vomiting prevents the taking of sufficient fluid rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose should be given every 4 to 6 hours With anuria the Borst regime (see p 481) may be used with a greater fluid intake in a tropical country The bowels are opened with an enema if there is constipation before the rectal salines are given A drip blood transfusion of 500 to 1 000 ml should be given to patients with polyuria Careful cross grouping is necessary to prevent hæmolytic reactions For peripheral circulatory failure treatment consists of warmth the intramuscular injection of 1 ml of Pitressin and the oral administration of ephedrine hydrochlor gr ½ repeated six hourly It is usually considered inadvisable to prescribe any anti malarial drug during the acute stages of the illness and the anæmia generally improves spontaneously during convalescence

After recovery it is advisable for the patient to leave a malarial country, as dangerous relapses are to be feared  Parasites *often reappear 5 to 14* days after the hæmoglobinuria has ceased and a course of chloroquin or amodiaquine should be given

## Bacillary Dysentery

### (*Epidemic Dysentery*)

**Definition** A disease characterised by tenesmus, diarrhœa with the passage of blood and mucus and caused by a special group of bacilli

**Etiology** There are three main types of bacilli  The Shigella shigæ (including the Shiga and Schmitz bacilli) which do not ferment manitol  the Shigella sonnei (Sonne bacillus), and the Shigella flexneri (including the Flexner and Boyd bacilli) which are manitol fermenting organisms  Flexner Boyd and Sonne infections are generally of a milder type than are those caused by the Shigella group  Infection is carried to man by food and water and transmitted from fæces by flies or by fingers  *Predisposing causes*  1 Locality  Bacillary dysentery occurs in epidemics in the sub tropics and also in temperate climates  Outbreaks of dysentery in England including summer diarrhœa in children are most often due to Salmonella infections to staphylococci to Shigella sonnei or to proteus morgani  It affects armies prisoners and lunatics in asylums  2 Season  In the tropics during the rainy season  3 Debility  4 Age  Infants under 2 years and adults of either sex

**Pathology** The bacilli produce an acute inflammation of the mucous membrane of the large intestine and the last part of *the ileum* may also be affected  Small superficial pinkish ulcers and larger irregular transversely disposed ulcers form and there is no undermining of their edges  Later the mucous membrane may necrose becoming *greenish black  This may slough and leave a firm rigid* tube of bowel

**Incubation Period** This varies from a few hours to about 7 days

**Clinical Findings** The patient is usually an adult who is suddenly taken ill with abdominal pain and diarrhœa  There is malaise associated with fever and tenesmus may be very marked  In an acute case the patient has to go to stool very frequently but little is passed each time  There may also be vomiting and considerable thirst

*On Examination* The abdomen is tender and the muscles are often held rather rigidly contracted  The temperature is raised to 102° F or 103° F and the pulse is frequent  The tongue in a severe case becomes dry and the patient suffers from marked collapse owing to dehydration and toxæmia  The urine output is scanty  The stools often consist of a little jelly like material with blood but with no fæcal matter and they are odourless  Microscopically a *film shows very few* bacilli many polymorphonuclear cells (up to 90%) some large macrophage cells with engulfed red corpuscles (these must be distinguished from amœbæ)  Dysentery bacilli may be isolated by culture *during* the first few days of the illness but rarely after the first week  The blood  A positive agglutination with stock dysentery

bacilli cultures can be obtained after the first week, most definitely in Shiga infections Blood culture is rarely positive there is usually a slight leucocytosis In severe cases the blood urea rises and the alkali reserve falls

*Varieties* 1 Fulminating dysentery This is usually a Shigella infection It may be a choleraic type with collapse, vomiting and diarrhœa, or a gangrenous type with severe toxæmia and abdominal pain 2 Mild type In Great Britain the disease is not usually severe and commonly is due to the Sh Sonnei Many persons excrete this organism without suffering from clinical dysentery In children the onset is often abrupt with abdominal pain or lower abdominal dis comfort one or two loose green motions a day without blood or mucus and with little constitutional disturbance 3 Chronic type This persists for over a month often with alternating constipation and diarrhœa 4 Infantile or summer diarrhœa Diarrhœa with blood and mucus in the stools may be due to a Sonne infection

**Differential Diagnosis** Bacillary dysentery differs clinically from amœbic dysentery in its more acute onset higher temperature greater degree of tenesmus and more profound collapse The stools differ in their appearance those of amœbic dysentery usually being very offensive and containing fæces They differ also in their cellular exudate The causative organism is also distinct Arthritis is a complication of bacillary dysentery and liver abscess of amœbic dysentery Other conditions which can be excluded clinically and bacteriologically are the enterica group infections food poisoning cholera and schistoso miasis Ulcerative colitis is in some cases due to bacillary dysentery Sigmoidoscopic examination is of value in the diagnosis of chronic dysentery The mucous membrane is red granular and bleeds easily and the bowel wall is rigid

**Course and Complications** In fulminating Shigella cases the course is rapidly progressive to death in acute cases the diarrhœa usually lasts for 7 to 10 days and the condition gradually improves as the motions become fæculent Relapses may occur, the stools containing blood and mucus and a condition of chronic dysentery becomes established Complications include arthritis parotitis conjunctivitis iridocyclitis, ascites and constriction of the intestine The arthritis usually affects large joints such as the knees there is clear fluid in the joint and recovery is the rule It may occur during the acute stage or during convalescence

**Prognosis** The average mortality with early and adequate sulphonamide or antibiotic treatment is now between 0 05 and 2% Fulminating cases die in a few days Shiga infections are more severe than other types

**Treatment** *Prophylactic* Endeavours should be made to prevent flies from having access to food fæces should be disposed of hygienic ally dysentery bacilli carriers should not be allowed to prepare food

*Curative* The patient must be kept warm in bed Hot applications to the abdomen such as turpentine stupes help to relieve pain If there is severe colicky pain an injection of morphin hydrochlor gr $\frac{1}{4}$, may be

given for the first 2 days *The Diet* During the first 24 hours only water or mineral waters should be allowed Subsequently albumin water, dextrose water thin arrowroot Brand's essence and jelly are given Sufficient fluid must be taken to produce a urinary output of about 2 pints in 24 hours The diet is gradually increased by the addition of citrated milk, milk jelly, custard, sago pudding a lightly boiled egg toast or rusks, etc

*Special Treatment* Terramycin or aureomycin may be given in doses of 2 G followed every 12 hours by 1 G for 4 doses The drawback to these drugs is that they may lead to granular proctitis or to staphylococcal enteritis Sulphonamide treatment has proved of inestimable value in the treatment of bacillary dysentery Sulphaguanidine Sulfasuxidine or Sulfathalidine are slowly absorbed from the intestines and do not give rise to renal complications The average dose of any of these sulphonamides for an adult is 3 5 G this is repeated every 4 6 or 8 hours according to the severity of the disease When the diarrhœa ceases the dose of the drug is tapered off, the total course not lasting longer than 14 days

If there is severe dehydration 1 000 to 1 500 ml of normal saline containing 5% dextrose should be given intravenously Tenesmus can be relieved by a small starch and opium enema (Tnc opii m 20 starch gr 60, water fl oz 2) If these measures fail a cæcostomy will give rest to the large intestine and also enable it to be washed out and it is successful in some cases

## Amœbiasis

*(Amœbic Dysentery Amœbic Hepatitis Pulmonary Amœbiasis)*

**Definition** Amœbiasis includes the diseases in man caused by infection with the Entamœba histolytica

**Etiology** The causative agent is the Entamœba histolytica This exists in an amœboid or vegetative form and as a cyst The amœba is actively motile has a clear ectoplasm and usually contains engulfed red cells The cyst contains four nuclei Man swallows the cysts in food or water In the intestine amœbæ develop some of these amœbæ form cysts which are excreted in the fæces The house fly probably carries the cysts to food and water or food may be contaminated by the fingers of a cyst passer *Predisposing causes* Locality especially Egypt India Mesopotamia and parts of America

**Pathology** The amœbæ infect the large intestine working their way through the mucous membrane They cause colliquative necrosis in the submucous tissues and a bottle shaped ulcer forms The ulcers enlarge and have an undermined edge They are situated longitudinally in the intestine and form especially in the cæcum and at intestinal flexures The amœbæ may pass to the liver *via* the portal vein and produce hepatitis or single or multiple abscesses the pus is pinkish brown (anchovy sauce) and usually sterile Amœbæ are found in the scrapings from the abscess wall The liver abscess may rupture into the lung, stomach duodenum colon peritoneum and rarely into

the pericardium Abscesses may form in the brain or spleen as the result of a systemic infection

**Incubation Period** In experimentally infected man this varies between 9 to 94 days

**Clinical Findings** The patient may be a child or an adult, who complains of diarrhœa coming on gradually with abdominal pain There is tenesmus if the lesions are situated low in the rectum

*On Examination* Tenderness may be elicited over the cæcum or transverse colon or in the left iliac region The temperature is usually not raised or only slightly so In this acute simple type the bowels are opened about 12 times in the 24 hours and the motions contain mucus pus dark blood and some fæces If untreated the condition usually remits after a few weeks but recurrences are common Examination of the stools The specimen should be sent to the laboratory directly it is passed and examined for amœbæ or cysts and if not found another specimen sent daily for 5 to 7 days The cellular content of the stools in amœbic dysentery shows a preponderance of mononuclear leucocytes or degenerating epithelial cells The field usually swarms with bacteria, and Charcot Leyden crystals are often present In chronic cases diagnosis may at times be made by sigmoidoscopy and finding amœbæ in scrapings from an ulcer wall

Other *varieties* include 1 *Acute gangrenous dysentery* A short fulminating illness in which hæmorrhage and perforation are liable to occur proving fatal in a few days 2 *Chronic dysentery* (*a*) with symptoms such as anæmia debility and recurrent attacks of diarrhœa (*b*) Latent Here the patient may pass cysts and have intestinal ulceration without symptoms In other cases abscesses form in the liver or elsewhere without any previous symptoms of dysentery 3 *Amœbic hepatitis* This may complicate a definite case of amœbic dysentery or develop insidiously *Acute hepatitis* When the liver is involved there is usually a rise of temperature and pain is felt over the liver or in the right shoulder On examination there is tenderness over the liver and the blood usually shows a leucocytosis before an abscess forms In other cases there exists a chronic hepatitis without pain or fever or dysenteric symptoms there is usually however a leucocytosis *The hepatic abscess* is often solitary and usually on the right side as it enlarges the patient complains of pain either local or referred to the shoulder malaise headache and rigors or sweats may occur The liver is acutely tender over the abscess and a fluctuating swelling may be found with some œdema of skin over it In other cases the abscess may form under the liver and not be palpable Multiple small abscesses in the liver are unusual There is severe constitutional disturbance and often slight icterus The condition is very serious

*Pulmonary amœbiasis* This may be primary or secondary the latter variety being more common In the primary form the amœbæ spread by the blood stream from the intestine Direct extension from an amœbic abscess in the liver is the cause of the secondary variety In primary amœbiasis the patient often has hæmoptysis with prolonged fever interrupted by periodical exacerbations and signs are found

suggesting pulmonary tuberculosis E histolytica cysts may be found in the stools and the blood may show an eosinophilia There may also be a history of dysentery In the secondary variety, signs of con solidation or of abscess formation are usually found in the right lower lobe Less often the hepatic abscess ruptures into the pleura forming an empyema Communication of a liver abscess with the lungs results in the characteristic " anchovy sauce " sputum

**Differential Diagnosis** Amœbic dysentery must be distinguished from bacillary dysentery Clinically the diseases are similar pathologically they are totally different The stool examination is the real criterion The non pathogenic amœba coli and its cysts must be differentiated from the entamœba histolytica The cysts of amœba coli contain eight nuclei The amœba coli does not engulf red cells, is very slightly motile and it has no clear ectoplasm Amœbic hepatitis may be mistaken for gall stones or for a suppurating hydatid cyst of the liver A history of dysentery is often not obtainable The response to emetine treatment and finding cysts in the fæces establish the diagnosis Pulmonary amœbiasis is most likely to be mistaken for pulmonary tuberculosis owing to the hæmoptysis or for other forms of lung abscess Here again the condition is often met with apart from any definite history of dysentery, and the diagnosis is established by the response to emetine treatment Thickening in the region of the cæcum may suggest a carcinoma

**Course and Complications** The course depends entirely on the treatment adequate early treatment cuts short the disease and prevents relapses some chronic and neglected cases are very difficult to cure Complications include Intestinal perforation intestinal stenosis with partial obstruction and gangrene hepatic abscess which may rupture into various sites cholecystitis pulmonary abscess cerebral abscess Abdominal adhesions are liable to follow a liver abscess which reaches the surface

**Prognosis** This is good in uncomplicated cases properly treated Brain abscess is probably always fatal Liver abscess with modern treatment has a low mortality Multiple liver abscesses are more grave Lung abscesses usually recover with treatment

**Treatment** *Prophylactic* Water and raw food should be protected from house flies Cyst passers should not be employed as cooks Cyst carriers may be treated with Diodoquin in tablet form 0 21 G 3 tablets every 8 hours for 2 weeks

*Curative* The patient with acute dysentery should be kept warm in bed The diet should be restricted to albumin water milk and dextrose orangeade in quantities of 2 to 3 pints a day Emetine hydro chloride gr 1 in 1 ml of distilled water should be injected subcu taneously or intramuscularly every evening for 5 to 10 doses The patient must stay in bed during this time owing to the cardiac depres sant effect of the emetine The diarrhœa usually ceases after 4 or 5 days A sterilising course of treatment should now be given as is also used for chronic cases Emetine bismuth iodide (E B I ) by mouth usually proves very efficacious E B I gr 2 is given in a hard gelatin capsule

at night 3½ hours after the last meal To prevent nausea and vomiting phenobarbitone gr 1½ should be administered by mouth 1 hour before the E B I This is given for 12 doses Primary pulmonary amœbiasis usually responds quickly if emetine hydrochloride gr ½ is injected subcutaneously twice daily and carbarsone 0 25 G in a gelatin capsule is given by mouth b i d p c for 5 days Carbarsone is then given alone for another 5 days If the condition has not resolved after 5 days freedom from the treatment the course is repeated Favourable results have been reported by treating amœbic colitis with aureomycin and terramycin mg 250 every 6 hours for 2 weeks After 2 weeks interval the course is repeated During convalescence the patient should avoid red meat spices rich food cheese and potatoes

Amœbic abscess of the liver is best treated by aspiration of the pus and a course of 12 subcutaneous injections of emetine or by the administration of chloroquine 4 tabs (= 600 mg base) once daily for 2 days and then 3 tabs once a day for 21 days Open operation should not be performed unless the pus is found to be secondarily infected or the abscess is in a position inaccessible for aspiration The mortality has been much lowered by closed aspiration treatment Emetine hydrochloride gr 1 in 1 oz distilled water may be injected into the abscess cavity after aspiration

## Cholera

Definition A disease due to a specific bacillus characterised by severe watery evacuations muscular cramps and collapse

Etiology The cause is the Vibrio choleræ (comma bacillus) It is conveyed from man to man by means of infected water and food such as melons milk etc The vibrios pass out in the fæces and the water is thus contaminated Fingers and flies may also convey the vibrios Carriers occur but they are usually convalescent patients who remain carriers for a few weeks or months only The disease was disseminated largely by pilgrims or traders *Predisposing causes* 1 Locality Cholera occurs chiefly in India endemically and epidemically Epidemics have arisen in Europe and America 2 Season and climate The incidence is favoured by a high absolute humidity of the air (over 0 400)

Pathology At autopsy the body is wasted and dehydrated the muscles are dark The chief lesion is in the ileum where the mucous membrane is reddened and the contents may be of the rice water character white flakes of cellular *débris* in a clear fluid The spleen is often small the kidneys may show areas of focal necrosis and hyaline changes in the convoluted tubules

Cholera vibrios are usually confined to the intestine but some may be found in the gall bladder more rarely there is a septicæmia and they are present in the spleen and urine

Incubation Period This varies from a few hours to 5 or 6 days

Clinical Findings Cholera may begin quite suddenly or there may be a few days of diarrhœa not of a typical choleraic nature at the onset The patient complains of very severe diarrhœa in which after the

bowel has been emptied of fæcal matter the evacuations consist mainly of water There is marked prostration, and severe cramps in the legs and abdomen, which *may cause muscle rupture* *Vomiting* of watery fluid adds to the patient s misery, and there is intense thirst

*On Examination* In this algid stage the skin is cold and clammy, and has lost its elasticity the patient is cyanosed, the eyes are sunken, the respirations and pulse rate are rapid, and the voice is feeble The axillary temperature is normal or subnormal, but in the rectum the temperature is slightly raised The stools are of a rice water" character (see above) and contain cholera vibrios Bacteriophage is present in stools of patients who do well, and not present in fatal cases The urine becomes very scanty with a high specific gravity and albumin is often present The blood pressure falls below 100 mm Hg syst The blood is sticky" from *concentration* the Hb *percentage rises* and the red cells number over 5 000 000 per c mm There is a leucocytosis of 20 000 to 50 000 per c mm The specific gravity of the blood rises from the normal of 1 056 to 1 061 if 1 pint of fluid has been drained away from the blood and to 1 065 with a loss of 5 pints The blood culture is usually sterile but a positive agglutination of dead cholera vibrios by the serum is found after the eighth to tenth days The patient may rapidly die, or in a few days pass into the reaction stage The body temperature rises the skin becomes warm the blood pressure rises the pulse slows and the output of urine increases the watery vomit and stools cease An irregular type of fever may now ensue with delirium and coma called cholera typhoid or the temperature may rapidly settle as the patient's condition improves

*Varieties* 1 *Cholera sicca* The patient rapidly dies the bowels contain much fluid but no rice water stools have been passed

2 *Ambulatory* Here the diarrhœa is not very severe the constitutional disturbance is comparatively mild and there are no cramps

*Differential Diagnosis* *There is little difficulty in an epidemic* and the diagnosis is made by the naked eye appearance of the 'rice water stools when they are placed in a test tube and by an examination for vibrios Other causes of acute gastro enteritis must be excluded such as food poisoning and in England cholera nostras or summer diarrhœa In tropical countries algid malaria is excluded by a blood film

Course and Complications The severe forms of cholera *take a very* rapid course in less grave infections the course is more prolonged and relapses may occur during convalescence with return of the diarrhœa Complications include Bronchopneumonia, parotitis, renal failure uræmia and hyperpyrexia

Prognosis Cholera is a very rare disease but the mortality has been lowered by modern treatment to an average of about *20 per cent* In any epidemic the case virulence becomes less severe towards its close The amount of fluid lost from the blood (as judged by the specific gravity) is a guide to the severity of the case

Treatment *Prophylactic* In an epidemic protection is afforded by boiling all water and milk eating only cooked foods avoiding all foods liable to cause diarrhœa, and by the isolation of contacts Further, at

any time vaccination will afford considerable protection for several months The anti cholera vaccine contains 8 000 million dead vibrios in 1 ml the initial dose is 0 5 ml followed by 1 ml 10 days later

*Curative* At the onset of the diarrhœa the patient should be kept warm in bed The fæces and urine should be received in disinfectant as for enteric fever Morphine and opium should never be used The *patient should drink water albumin or dextrose water in small quantities frequently* taking as much as he can Vomiting may be checked by giving tab cocain gr $\frac{1}{10}$ by mouth repeated in half an hour if necessary Rogers hypertonic saline should be given intravenously in all cases in which there is much loss of fluid from the blood, as judged by the specific gravity or in which the blood pressure falls to 80 or 70 mm Hg syst The chlorides combine with the cholera toxins in the blood and the product is excreted The hypertonic solution prevents fluid passing from the blood to the bowel Uræmic symptoms are checked by maintaining the alkalinity of the blood by intravenous injection of an alkaline solution Rogers' hypertonic saline solution contains sod chlorid gr 120 calc chlorid gr 4 water 1 pint and his alkaline solution contains sod bicarb gr 160 sod chlorid gr 90 water 1 pint The solutions should be warmed to body temperature but if the rectal temperature *is over 100°F the solutions should not be over 80°F or hyper* pyrexia may ensue They should be run in at the rate of about 4 oz a minute The amount required is determined by the specific gravity of the blood as described on p 726 One pint of the alkaline solution is first injected and then the requisite amount of fluid is made up by injecting the hypertonic solution The alkaline solution should also be injected into the rectum at first 10 oz every 2 hours and then every 4 hours until the output of urine exceeds 20 oz a day Sulphonamides and antibiotics are not of clinical value in cholera During convalescence the diet must be very cautiously increased with diluted citrated milk milk jellies custards etc

## Sprue

### (*Psilosis*)

*Definition A disease characterised by emaciation sore tongue and* diarrhœa

Etiology The cause is unknown Sprue may be due to 1 Damage to the villi of the small intestine preventing phosphorylation of fats glucose and galactose and diminished absorption of fats sugars and vitamins 2 Infection with yeasts such as the Monilia psilosis or with streptococci These are probably secondary infections 3 Deficiency of the vitamin complex B in the food *Predisposing causes* 1 Locality Especially China the Malayan Archipelago India Ceylon the East and West Indies and the southern states of N America Sprue may occur in certain houses 2 Race Europeans are liable to sprue in endemic zones and also may develop it several years after returning home 3 Age and sex Chiefly adults and slightly more common in women

Pathology The wall of the small intestine is very thin and the mucous membrane is atrophied Small ulcers may form and perforate

The large intestine may be similarly affected The heart, liver and spleen are atrophied and the bone marrow shows megaloblastic hyperplasia The atrophied *intestinal mucous membrane interferes with absorption*

**Clinical Findings** The onset is insidious with weakness, dyspepsia, a burning pain behind the sternum and flatulence causing abdominal distention Looseness of the bowels is then noticed The stools are frothy copious and offensive The patient loses weight progressively and complains of soreness of the tongue and mouth and later soreness may also be referred to the œsophagus and rectum

*On Examination* The *patient is very wasted when the disease is of* some duration and the skin is often dark the areas of pigmentation often occurring on the face The tongue in the early stages is red, and later small vesicles may form and the surface is smooth and shiny In more chronic cases the tongue is shrunken and pale Erosions may also be seen on the buccal mucous membrane Skin changes also include follicular hyperkeratosis due to deficiency of vitamin A and para keratosis from lack of vitamin B The liver dulness is diminished Signs of subacute combined degeneration of the cord are rarely found A test meal often shows deficiency of hydrochloric acid or a histamine resistant achlorhydria The barium meal may show clumping of the barium in the jejunum the so called 'moulage' sign The stools are typically large pale and frothy The pallor of the stool is not due to the excess of fat but to alteration of stercobilin to leucobilin the fat content may not be raised in a pale stool and conversely a coloured stool may contain an excess of fat In some cases the stools are copious and watery The total fat is usually increased to 50 or 80% (normal 20 to 30% of dried fæces) there being an increase of the split fat The blood *shows often a megalocytic anæmia with a high* colour index A few normoblasts may be seen, but usually no mego blasts In some cases there is an iron deficiency as shown by a low mean corpuscular hæmoglobin concentration The leucocytes are often below normal in number with a relative lymphocytosis, Lack of absorption of vitamin K may cause a hypoprothrombinæmia The dextrose tolerance test often shows a low curve due to deficiency of absorption and the blood calcium may be below normal Sternal puncture shows changes similar to those found in pernicious anæmia (see p 515)

**Differential Diagnosis** The history of residence abroad and the clinical picture is characteristic Cœliac disease in adults (see p 65) closely resembles and *probably is identical with sprue* *In chronic* pancreatitis there is an excess of unsplit fat In some cases the blood count may closely resemble that of pernicious anæmia

"Hill diarrhœa is liable to occur especially in India at an altitude of over 6 000 feet It resembles sprue with the pale frothy *stools* occurring in the morning and may develop into true sprue

**Course and Complications** The course is usually chronic but acute relapses may occur with the passage of 15 to 20 stools a day Hæmatemesis or tetany may occur as complications

**Prognosis** Death may occur within a year if the disease is not treated and in any case the prognosis is bad in patients past middle age

**Treatment** The patient must be put to bed and kept there for at least 6 weeks The diet consists of milk Benger s food or Yoghurt given in two hourly feeds beginning with 3 pints of milk in the 24 hours and increasing gradually to 5 pints It should be sipped very slowly Sprulac is rich in protein and poor in fat and is of value as an alternative to a milk diet It is dissolved in water according to the directions and six feeds are given in the 24 hours at intervals of 2 hours the total quantity taken being gradually increased from two to six pints If milk food disagrees raw meat juice or 2 oz feeds of lightly cooked minced steak may be given The meat feeds are increased up to 1 to 2 lbs daily The motions should become formed after a few days treatment If constipation develops liquid paraffin should be given as required After 6 weeks the diet should be gradually increased by adding fruit such as strawberries bananas or apples 1 or 2 daily and gradually increasing to 1 lb or more daily A raw egg may be added to the milk then 1 or 2 rusks pounded fish and later chicken are given Bism salicyl gr 15 t d s two hours after meals helps to check loose motions For the acute relapses Sulphaguanidine should be given in doses similar to those prescribed in bacillary dysentery

Liver extract or vitamin $B_1$ in doses similar to those given for pernicious anæmia are useful both to control the sprue and to cure the anæmia Folic acid may be given as an alternative to liver extracts The dose is mg 10 to 20 daily by mouth in the acute stage followed by mg 2 5 to 10 daily until cure is effected The disadvantage of this treatment is that neurological complications may develop and it should not be given if there is evidence of postero lateral sclerosis or of neuritis Tab Fersolate 2 t d s p c should be given if there is iron deficiency If the anæmia is very severe blood transfusion is of great value Nicotinic acid mg 100 t d s and riboflavin mg 3 t d s should be given for the stomatitis Deficiency of ionised calcium is made good by calcium lactate gr 15 to 60 t d s together with Radiostoleum capsules m. 3 t d s

After each feed the mouth should be cleansed with an alkaline solution containing sod bicarb gr 60 to 5 oz of water As soon as the patient is convalescent he should leave the affected area and not return

## Plague

*(The Black Death)*

**Definition** A disease characterised by septicæmia, and frequently by enlargement of a group of glands, due to infection with a specific bacillus

**Etiology** The cause is the Pasteurella pestis Rats suffer from plague and bubonic plague is conveyed to man by the bite of the rat flea (especially the *Xenopsylla cheopis*) Direct droplet infection from man to man causes the spread of pneumonic plague *Predisposing causes* 1 Locality Plague is endemic in parts of India Indo China the East Indies Siberia North and East Africa etc and

sporadic cases occur in Europe and in ports such as London In 1664–1665 the great epidemic occurred in London 2 Climate and season A mean temperature over 80° F is unfavourable to plague In temperate zones it occurs in the summer and autumn 3 Hygienic conditions Dirt and overcrowding favour its development 4 Age and sex No age or sex is exempt

**Pathology** The disease is a hæmorrhagic septicæmia Post mortem the body may appear livid (black death) owing to ecchymoses The lymph glands at various sites are enlarged matted together and surrounded with a hæmorrhagic œdema Serous membranes show ecchymoses and blood stained effusions The spleen is enlarged and soft, the liver is enlarged, due to cloudy swelling and small abscesses may be present the kidneys may show cloudy swelling, ulcers may be found in the colon The lungs may show hæmorrhagic broncho pneumonic patches or lobar involvement The causative organism may be isolated from the blood, lymph glands spleen lungs, etc

**Incubation Period** 2 to 10 days

**Clinical Findings** In bubonic plague the patient is suddenly taken ill with malaise shivering pains in the back and legs nausea and vomiting He becomes very weak is mentally confused and the gait may be staggering In a day or so he feels a painful swelling in a groin or elsewhere owing to the bubo

*On Examination* The face is dusky or flushed the conjunctivæ injected and the expression may be wild The temperature is raised, usually over 102° F and the pulse frequent The primary bubo is generally in the groin the glands being enlarged, tender and the subcutaneous tissues over them feeling œdematous Less often the primary glandular enlargement occurs in the axilla or neck Fluid obtained by gland puncture shows plague bacilli The blood There is a high leucocytosis which may reach 80 000 per c mm The blood culture may show the Past pestis The urine often contains albumin The glands usually, but not invariably suppurate in about 7 to 10 days The temperature runs an irregular course and in cases which recover it falls by lysis when the glands if they do not suppurate, diminish in size and often there is marked sweating Skin lesions may occur ecchymoses are common but in addition there may be vesicles, pustules or areas of gangrene resembling carbuncles

*Varieties* 1 *Abortive plague* (*Pestis minor*) Pyrexia may be slight or absent and the enlarged glands are not very painful Constitutional disturbance is slight and the patient remains ambulatory The buboes may or may not suppurate

2 *Pneumonic plague* This may be primary or complicate bubonic plague The signs and symptoms are pulmonary there is marked dyspnœa with cough and expectoration

*On Examination* The patient is dusky and cyanosed râles may be heard scattered in the lungs or areas of bronchial breathing with increased voice conduction A pleural effusion often forms The sputum is thin, watery and coloured pink with blood, it is not sticky and contains the Past pestis

3 *Septicæmic plague* The patient usually dies before a bubo or bronchopneumonia has had time to be manifest

4 *Intestinal plague* This is an unusual variety in which buboes do not form, but there is diarrhœa with blood and vomiting Plague bacilli are found in the stools

5 *Cerebral plague* This is also a rare variety characterised by convulsions and coma

**Differential Diagnosis** In an epidemic there is little difficulty in recognising plague Sporadic cases may be confused with other forms of septicæmia with enteric fever glandular fever malaria (cerebral) or influenzal bronchopneumonia The buboes must be distinguished from those due to lymphogranuloma venereum and tularæmia Diagnosis is established by finding the causative organism in the fluid from gland punctures the blood urine fæces or sputum

**Course and Complications** The course of bubonic plague is as described above the course may be rapidly fatal in the pneumonic septicæmic or intestinal type and this is the rule with cerebral plague Complications include pneumonia in bubonic plague and severe hæmorrhage from erosion due to a sloughing bubo

**Prognosis** This has been considerably improved by the use of sulphonamide drugs and antibiotics The mortality for the pneumonic and septicæmic varieties was formerly nearly 100% the bubonic variety proved fatal in about 80% of cases in natives and in about 30% of cases in Europeans

**Treatment** *Prophylactic* This is chiefly concerned with the destruction of rats and prevention of rats from entering houses or ships Nurses should wear masks in caring for cases of pneumonic plague In an epidemic a vaccine containing dead bacilli should be given 1 000 millions for the first dose and 2 000 millions 7 days later It appears to exert definite protection

*Curative* The patient should be isolated for 4 weeks after the temperature is normal He must be kept in bed and the temperature lowered by sponging if it rises over 106 F The buboes should be fomented every 4 hours with 1 in 4 000 perchloride of mercury solution When the buboes suppurate they should be opened Sulphadiazine should be given first 4 G followed by 2 G every 4 hours by day and night until the temperature is normal In severe cases the dose should be doubled for the first 24 hours When the temperature is normal 0 5 G should be given every 4 hours for 14 days The urine must be kept alkaline during the treatment and at least 4 pints of fluid taken every 24 hours Streptomycin 0 5 G may be given intramuscularly every 6 hours together with the sulphadiazine In every case the urine and fæces should be disinfected as for typhoid fever

## Yellow Fever

**Definition** An acute disease characterised by fever jaundice albuminuria and hæmatemesis

**Etiology** Yellow fever is caused by an ultra microscopic filterable virus In the urban and rural type of the disease the virus is

*conveyed from man to man*, or from animals such as monkeys to man by a mosquito the *Aedes ægypti* (*Stegomyia fasciata*) Patients are infective to mosquitoes for the first 4 days of their illness The virus *undergoes changes in the mosquito so that the latter* does not become capable of infecting man for 10 to 12 days but remains infective for its lifetime (about 2 months) Indian monkeys can be infected by the blood of a patient but West *African monkeys are less susceptible* The serum of a patient who has recovered from yellow fever protects a susceptible monkey from the virus Jungle yellow fever occurs in Brazil, and man is infected by various forest *mosquitoes* *It is probable that* monkeys and other forest animals act as reservoirs of infection for urban and jungle fever *Predisposing causes* 1 Locality Yellow fever is endemic in West Africa and to a lesser degree in Brazil and Mexico It was formerly very prevalent in Panama and Havana 2 Climate A mean average temperature of over 75 F is necessary, and preferably a moist heat

Pathology The skin is very yellow Hæmorrhages may be seen under the skin in the muscles stomach intestines, pleuræ meninges kidneys and liver The liver is soft and yellow and shows mid zonal necrosis The necrotic hyaline cells are known as Councilman bodies Characteristic intranuclear inclusion bodies are seen in the liver cells The spleen appears normal The kidneys show fatty changes, and casts may be found in the convoluted tubules

Incubation Period This is usually 3 to 6 days

Clinical Findings In the average severe type of the disease, the onset is sudden with a rigor and rise of temperature *The disease may* fall into 3 stages *The initial fever* This lasts 3 or 4 days The patient complains of severe headache pains in the eyes back and the *calves of the legs He may also have epigastric pain and vomiting*, with constipation and insomnia The virus can be isolated from the blood during the first 3 days of the fever

*On Examination* The face is flushed, the eyes bright, the skin dry the pulse is somewhat rapid, about 120 and full The urine diminishes and albumin appears in increasing quantities Jaundice appears about the third day The vomit at this stage usually contains bile but some blood may be present

*The period of remission* This lasts 2 or 3 days There is some improvement in the general condition of the patient The temperature falls and the pulse becomes slow The tongue is furred but the tip and edges are clean Convalescence may now ensue but usually the patient passes into *the third stage of secondary fever* The temperature rises again but the pulse rate remains slow (*Faget s sign*) The jaundice increases, the output of urine diminishes much albumin is present with granular casts, red blood cells hæmoglobin and bile ' Black vomit ' *occurs* which is due to altered blood and there may be diarrhœa with melæna There are often hæmorrhages from the nose gums and into the skin Death may follow or the patient may gradually recover

*Varieties* 1 *Mild cases* There may be only slight fever with headache and a little vomiting, and the patient is well in a few *days*

2 *Malignant cases* The temperature rapidly rises to over 106° F with severe vomiting of blood suppression of urine coma and death

**Differential Diagnosis** The characteristic features of yellow fever are the jaundice black vomit massive albuminuria and slow pulse, with rising temperature Mild cases may resemble dengue but in the latter a rash is usually seen about the fifth day Weil s disease may be hard to differentiate clinically but it occurs in the absence of the mosquito which transmits yellow fever The diagnosis of Weil s disease can be established by special laboratory tests In malaria the spleen is usually enlarged and parasites are found in the blood

**Course and Complications** The course is as described above Complications include Abscesses in the skin parotitis and occasionally relapse

**Prognosis** The average mortality varies between 20 and 50% *The outlook is unfavourable if the temperature rises to over 105 F or* if there is suppression of urine

**Treatment** *Prophylactic* The disease has been stamped out in Panama and other places by mosquito destruction Water tanks should be protected by fine metal gauze covers and certain small fish may also be placed in the water to eat up the mosquito larvæ Mosquito nets should be used at night Mosquito control is not applicable for controlling jungle yellow fever Prophylactic inoculation with attenuated yellow fever virus in the preparation of which human serum is not used as this led to the occurrence of post vaccination jaundice is now being practised with success especially in the jungle type of fever The dose is 0 5 ml It must not be given during the period 2 weeks before and 3 weeks after a primary smallpox vaccination Vaccination gives immunity for 4 years

*Curative* There is no specific cure known The patient should be put to bed and kept warm Barley water alkalis and dextrose should be given by mouth in the form of dextrose orangeade or lemonade (8 oz of dextrose in 2 quarts of water with the juice of 2 oranges or lemons) 3 to 4 pints daily and about gr 180 of sodium bicarbonate in the 24 hours sufficient to render the urine alkaline The diet must not be increased during the period of remission except to give half ounce feeds of half strength milk and lime water *During* convalescence only easily digested foods such as eggs in milk soups etc should be given Aperients should not be given by mouth after the onset of the illness enemata should be used if required The vomiting may be relieved by giving small drinks of iced champagne or drop doses of liq iodi mitis m 2 in 1 oz of water every hour and by applying a mustard leaf over the epigastrium If the dextrose cannot be taken by mouth rectal salines containing 5% dextrose may be given in doses of 6 to 8 oz every 4 hours or an intravenous injection of 10 oz of normal saline containing 5% dextrose

## Leprosy

**Definition** A disease characterised by the formation of specific infective granulomata affecting chiefly the skin and subcutaneous tissues mucous membranes and peripheral nerves

**Etiology** Leprosy is caused by the Mycobacterium lepræ (Hansen's bacillus) It is thought that infection occurs in the majority of cases through the skin less frequently by inhalation No intermediate agent is known The disease is thus probably spread by direct contact with an "open" case of leprosy, which *may not have been diagnosed* Nodular cases are more infectious than nervous ones Children born of lepers develop the disease in over 40% of cases unless removed from their parents at birth the disease is not hereditary *Predisposing causes* 1 Locality Leprosy is endemic in various parts of the world especially in India China Japan tropical Africa the East and West Indies, cases also occur in Europe, *e g* in Iceland Norway the Balkan States and in Australia and America 2 Unhygienic home conditions and overcrowding 3 Age The greatest number of cases show themselves between the ages of 10 and 30 years

**Pathology** Granulomata are produced containing "lepra cells" which may enclose lepra bacilli The bacilli are found in the skin nodules and ulcers in the nasal mucous membrane in the liver and spleen, in the blood at times during life and to a lesser extent in the nerves

**Incubation Period** In the majority of cases this is between 2 and 5 years

**Clinical Findings** The onset is insidious and before any lesion is apparent there may be prodromal symptoms such as malaise, muscular pains sweating with some pyrexia lasting for 7 to 10 days at a time There are three main types which will be described separately

1 *Lepromatous* (nodular) When cases are carefully observed the initial lesion is often found to be solitary It is situated usually on the buttocks or back or *extensor surface of limbs or on the cheek* The lesion is a small red or reddish brown slightly raised shiny spot about 0 5 cm in diameter further crops of spots appear with fever and on fading they may leave some pigmentation Some of the spots do not disappear but enlarge to form nodules and these may ulcerate Thicken*ing of the skin and face and ear lobes appears* which together with the nodules causes a leonine appearance the outer part of the eyebrows falls out Nodules may form on the conjunctiva or in the mouth or larynx and by ulceration cause blindness hoarseness etc *Necrosis of* deeper tissues may occur with loss of fingers or toes

2 *Neural* (maculo anæsthetic) In this variety macules may be seen on the skin in the early stages, 1 or 2 inches in diameter and of varying colour—pink violet, brown or white Sensation to light touch is often absent over these areas and *they do not sweat* Thickening also occurs in nerves the ulnar nerve becomes palpable at the elbow and other nerves such as the peroneal or tibial may be felt Disturbance of their function results in loss of sensation to touch heat cold and pain and muscular wasting especially of the small muscles of the hand with a claw deformity The most important sensory test is thermal anæsthesia The lesions may result in a bilateral facial paralysis Perforating ulcers occur in the feet

3 *Indeterminate* (mixed) A case may begin *as nodular* leprosy and

later nerve changes occur or more rarely *vice versâ*, or both types may develop simultaneously

In 1948 a classification based on the histopathology of the disease was introduced The types described are lepromatous tuberculoid and indeterminate

**Differential Diagnosis** Diagnosis is made in early cases by the appearance of the lesions and by finding the lepra bacilli This is effected by cutting out a small portion of the skin and making a smear on a slide from the subcutaneous tissue or by examining juice obtained from a nodule or by nasal scrapings The organism must be differentiated from the tubercle bacillus The differential diagnosis includes other skin diseases such as erythema multiforme lupus vulgaris mycosis fungoides syphilis and nerve lesions such as syringo myelia The histamine reaction consists in pricking the skin through a drop of 1/1 000 histamine phosphate In leprosy a wheal forms but there is no surrounding erythema

**Course and Complications** The course of an untreated case is usually prolonged for 10 to 20 years or more The disease passes through phases such as the early quiescent stage during which there is a slow multiplica tion of the bacilli with spread of local lesions this is followed by a reactionary stage with rapid proliferation of the bacilli and inflammation of the lesions and constitutional disturbance, and later there is a resolu tion stage in which the bacilli become granular and break down and the local lesions tend to heal The patient may then present marked deformities but the disease is arrested The most important com plications are pulmonary tuberculosis and nephritis

**Prognosis** The use of the sulphone drugs has considerably improved the prognosis

**Treatment** *Prophylactic* The incidence of leprosy can be materially lowered by 1 Isolation of active cases 2 Examination of home contacts every 6 months for 5 years 3 Removal of the children of lepers at birth 4 Treatment of early cases

*Curative* Early cases if not infective can be treated in clinics more advanced and infective cases should be isolated Best results are obtained from the use of Promanide (promin) Diasone or Sulph etrone Promin is given intravenously in doses of 2 to 5 G for 6 days and after a day s rest for another 6 days After a week the course is repeated and so on for several months Diasone is given by mouth as a 0 3 G tab 1 to 3 daily with meals After 10 weeks treatment the drug is discontinued for 2 weeks and then another course is given and so on Sulphetrone is given similarly but the tablets are 1 G Each tablet is crushed and mixed with gr 10 of sod bicarbonate During treatment the blood count should be done and iron and yeast given by mouth The use of chaulmoogra oil is gradually being abandoned

## Relapsing Fever

(*Famine Fever* *Tick Fever*)

**Definition** An intermittent fever due to a treponeme

**Etiology** Relapsing fever is caused by a treponeme of which

there are several varieties morphologically similar but which can be differentiated serologically The average length of the treponeme is 18μ The following are the main types The Treponema recurrentis (spirillum obermeieri) This is conveyed to man by the bites of lice (*Pediculus vestimenti* and *capitis*) The treponemes may be excreted in the fæces of the louse or exuded from its body fluid if it is crushed on the skin and they are inoculated usually by scratching The treponemes are found in the blood of the patient during the febrile periods of the disease and lice become infective about 16 days after biting such a patient The type of relapsing fever thus produced is met with chiefly in Eastern Europe and occasionally in North and West Africa, India, Ireland and America Epidemics occur chiefly in the winter especially amongst the poor Treponema duttoni This is conveyed by a tick (*Ornithodorus moubata*) These ticks infest houses and there is no seasonal incidence of the disease The treponeme causes African tick fever especially in Eastern and Central Africa and the Congo Other varieties include The T carteri (India) T berberum (N Africa) conveyed by lice and T persicum (Persia) conveyed by ticks

**Pathology** Post mortem the skin is often yellow and petechiæ may be seen The spleen is enlarged and may contain infarcts Treponemes are found in the spleen liver and bone marrow

**Incubation Period** 2 to 10 days

**Clinical Findings** The onset is usually sudden with shivering or a rigor severe headache, pains in the eyes legs and wrists giddiness, nausea vomiting and abdominal pain There may also be epistaxis

*On Examination* The temperature rises rapidly to 104° F or over and the pulse is frequent There may be slight icterus of the con junctivæ or definite jaundice and in some cases a pink macular rash is seen on the neck trunk and limbs with some petechial hæmorrhages The spleen is enlarged and there may be tenderness over the liver The blood Treponemes are present There is usually a leucocytosis and the Wassermann reaction may be positive The adhesion test is also positive, specific immune serum causing the treponemes to adhere to platelets The temperature remains irregularly raised for about 4 to 6 days and then falls by crisis with sweating For the next week the patient feels well and is afebrile A relapse occurs with a sudden rise of temperature and a return of symptoms lasting for about 2 days Convalescence then generally ensues

*Varieties* African tick fever The onset is usually more gradual relapses more numerous and the pyrexial period is shorter 1 to 2 days

**Differential Diagnosis** At the onset the disease may be mistaken for such conditions as influenza, malaria dengue or typhus fever Diagnosis is established by finding the treponemes in the blood

**Course and Complications** The course varies somewhat with the different types Complications include Pneumonia otitis parotitis nephritis hæmatemesis and meningeal symptoms

**Prognosis** The disease is a serious one unless adequately treated The African variety is usually more fatal than that caused by the T recurrentis

Treatment *Prophylactic* Lice in the clothes and head should be destroyed and native houses avoided which are infested by ticks

*Curative* The patient must be put to bed, kept on a fluid diet the bowels opened with a saline, and an intravenous injection given of neoarsphenamine 0 4 G for a man 0 3 G for a woman, and 0 1 G for a child It is most effective if injected as the temperature is rising and should not be given immediately before the crisis is expected or severe collapse may result One injection is usually sufficient to prevent relapses If arsenic is contra indicated owing to liver or kidney damage or a hæmorrhagic tendency penicillin should be injected 1 200 000 units of a procaine preparation every 12 hours for 4 days

## Scrub Typhus

**Etiology** Scrub typhus is caused by the Rickettsia tsutsugamushi (R orientalis) The vector is a mite the Trombicula akamushi or other trombiculid mites The mite only bites once and when thus infected with blood by biting an infected rodent transmits the infection to its offspring which now can infect man or an animal by its bite The mites are most plentiful in damp vegetative areas Rats and mice are the chief reservoirs of infection Locality South and East Asia North Australia and the Pacific Isles

**Pathology** The rickettsiæ multiply in the vascular endothelial cells Deficient blood supply to the central nervous system is believed to account for the nervous and for some of the circulatory disturbances

**Incubation Period** 7 to 21 days

**Clinical Findings** Scrub typhus was an important cause of sickness amongst troops in the 1939-45 war Previously most of the endemic *areas were not densely populated The clinical features resemble those* of louse borne typhus fever The onset is usually sudden with a chill *headache and conjunctivitis A primary eschar may be found in over* 50% of cases in the axilla perineum groin or trunk etc There is *generalised lymphadenitis towards the end of the first week A maculo* papular rash is seen about the fourth day on the trunk face and proximal part of the limbs *Mental symptoms predominate during the* second week with a fall of blood pressure and temporary deafness is not uncommon The white cells in the blood are not generally *increased in* number In over 50% of cases the spleen is palpable The temperature remains raised with daily remissions for about 2 weeks and then falls by lysis In scrub typhus the blood shows a modification of the Weil Felix reaction There is an agglutination of between 1/250 and 1/12 500 with Proteus OXK and a feeble agglutination with OX19 Proteus OXK is a strain of B proteus

**Differential Diagnosis** Malaria and dengue may be mistaken for scrub typhus and malaria and scrub typhus may coexist If the scar of the mite bite is visible the diagnosis can be made with more certainty at the onset Flea borne typhus may occur in the tropics but louse borne typhus is very rare there

**Course and Complications** Convalescence is usually slow and

the patient is likely to be off duty for 3 months or longer Bronchitis and bronchopneumonia may occur as complications

**Prognosis** This has been much improved with antibiotic treatment

**Treatment** *Prophylactic* Vaccine treatment has not proved successful Clothing can be smeared with dimethyl phthalate, which is a mite repellent Camps should be placed in dry sites and cleared of surrounding vegetations

*Curative* This is as for louse borne typhus Chloramphenicol aureomycin or terramycin should be given in doses of 3 G, followed by 0 25 G every 3 hours until the patient is afebrile Penicillin may be of value in cases complicated by bronchopneumonia, given by intra muscular injection of 600 000 units of a procaine preparation every 12 hours for 7 to 10 days

## Trypanosomiasis

### (*Sleeping Sickness*)

**Definition** A disease characterised by enlargement of lymphatic glands, followed by nervous changes and increasing torpor

**Etiology** Sleeping sickness is due to infection with a trypanosome, a flagellate protozoon Man is infected by the bite of a tsetse fly There are two main types T gambiense conveyed by the fly *glossina palpalis* occurring in Uganda and the Congo T rhodesiense conveyed by the fly *glossina morsitans* occurring in Rhodesia Nyasaland Tanganyika and Kenya In tropical America there is a variety caused by the T cruzi The trypanosome undergoes a cycle of development in the tsetse fly and finally reaches the salivary glands to infect man when he is bitten The fly may become infected by biting infected big game or man Rarely the fly will bite a man and carry the infection direct to another man, without any intermediate cycle *Predisposing causes* 1 Locality The disease is endemic in Equatorial Africa and America T gambiense infections occur near water and T rhodesiense in dry areas 2 Age Children and adults are infected 3 Race White and black races are equally susceptible

**Pathology** Post mortem there may be macroscopical evidence of meningitis or changes only seen microscopically in the meninges over the brain and cord Meningo encephalitis and meningo myelitis may also occur with perivascular infiltration of lymphocytes The changes resemble those found in dementia paralytica The cerebro spinal fluid is under pressure and trypanosomes occur in it Lymphatic glands are enlarged

**Incubation Period** This is variable, 2 weeks or longer

**Clinical Findings** The illness begins insidiously, the patient feeling unwell owing to fever

*On Examination* During the first stage, the temperature is irregularly raised, the pulse is frequent the posterior cervical glands are enlarged there may be some œdema of the legs and in white people a circinate erythema may be seen on the trunk The blood An excess of globulin may be demonstrated in the serum Trypanosomes may be found in the deposit after centrifugalisation of the blood The glands

Examination of the juice removed by puncture is more likely to show trypanosomes than is a blood film The disease, if untreated gradually *passes after months or years into the second stage characterised by* changes in the cerebro spinal fluid These include an excess of cells (50 or more per c mm ) and excess of protein (over 0 05%) Trypanosomes are not often demonstrable The patient becomes sluggish mentally and physically he appears dejected complains of pains in the body and loss of strength The glands in various parts of the body become larger He becomes more sleepy and dozes during the day Tremors are seen in the tongue and muscles of the arms The blood now shows an anæmia, and the large mononuclears may be increased to about 10% or over In the third stage the patient is bed ridden and finally becomes comatose

Differential Diagnosis Malaria is excluded by blood examination and the failure to respond to quinine or other anti malarial drug Finding the trypanosomes by gland puncture usually establishes the diagnosis in the early stages

Course and Complications The course of an untreated case is as described above the disease can now usually be arrested by early treatment Complications include Septic rhinitis otitis terminal pneumonia and dysentery

Prognosis This depends largely on early treatment but infections with the T rhodesiense are more grave than those with the T gambiense

Treatment *Prophylactic* It has been suggested that big game should be destroyed in endemic zones as they harbour the trypanosomes They may however be beneficial the tsetse flies biting them rather than man The *glossina palpalis* lives in vegetation near water and trees should be cleared in these zones Travellers through such districts should wear gloves and helmets Spread of the disease can be checked by the compulsory treatment of all natives affected D D T powder *sprayed from aeroplanes as for the prophylaxis of malaria is effective* in destroying the tsetse fly

*Curative* Early cases may be treated by the intramuscular or intravenous injection of Pentamidine given as a 10% solution The dose is mg 100 intravenously daily for 10 to 20 days or mg 100 intramuscularly the first day and mg 200 daily subsequently for 9 days Anthisan mg 100 tab should be given half an hour before the injection to prevent unpleasant symptoms due to flushing and fall of blood pressure Tryparsamide yields the best results in more advanced cases It is given intravenously or intramuscularly 1 to 3 G in 10 ml of water every week until a total of 20 to 100 G has been given according to the severity of the disease The average dose is reckoned on a basis of 0 04 G /kg of body weight Visual disturbances are a signal for stopping the injections as optic atrophy may ensue Mel B may be used for advanced cases which are resistant to tryparsamide It is a compound of melarsen oxide and dimercaprol and contains trivalent arsenic The dose is mg 3 6/kg body weight dissolved in propylene glycol and injected intravenously for 4 days It is toxic and should only be used in hospital

## Dengue

### (*Break bone Fever*)

**Definition** A disease characterised by fever and severe pains in the back and limbs

**Etiology** The causative agent is an ultra microscopic and filterable virus which is present in the blood It is conveyed from man to man by the mosquito *Aedes ægypti* (*Stegomyia fasciata*) The virus undergoes some development in the mosquito the latter becoming infective in 11 days The patient s blood is infective for the mosquito during the first 2 days of the illness Large epidemics and sporadic cases occur *Predisposing causes* 1 Locality The disease is met with in New South Wales, Florida Brazil the West Indies Fiji, Syria, Greece, Turkey and Africa 2 Climate Heat and moisture are required A previous attack usually confers immunity

**Pathology** The disease is rarely fatal, but œdema of the lungs has been noted

**Incubation Period** This is usually 4 to 10 days

**Clinical Findings** Three stages are described *The invasion* The onset is usually sudden with a rigor The patient complains of aching in the head and eyes and excruciating pains in the lower part of the back and legs which cause complete prostration

*On Examination* The conjunctivæ are injected, the face is flushed the skin generally hot and dry with some erythema The temperature rises rapidly to 102° F or higher The blood shows a leucopenia of about 2 000 white cells per c mm owing to diminution of the polymorpho nuclears which may fall to 40 or 50% The urine often contains a trace of albumin

*The remission* About the third day sweating occurs with often nose bleeding and diarrhœa the temperature falls by lysis or crisis and the patient feels better *Terminal fever* About the fifth day the temperature rises again to 100° F or so and remains raised for about 24 hours There is recurrence of the pains and a rash appears This is usually morbilliform in character and starts on the palms and backs of the hands spreading to arms, trunk and legs It fades in 2 or 3 days with slight desquamation

**Differential Diagnosis** The occurrence of the disease with its typical course in a warm climate is characteristic It must be diagnosed from other conditions such as influenza acute rheumatism malaria and measles

**Course and Complications** The course is usually as described Complications include Epistaxis bleeding from the gastro intestinal tract or uterus boils periarthritis of the knees or ankles and post dengue debility

**Prognosis** This is good The disease is practically never fatal

**Treatment** *Prophylactic* This is concerned with elimination of mosquitoes and prevention of their bites as for yellow fever

*Curative* The patient should be kept in bed until the rash has gone A saline aperient should be given at the onset Drugs are required for

the relief of pain aspirin in doses of gr 10 six hourly, tab codein co 1 six hourly, and in some cases a subcutaneous injection of morphin sulph gr 1/6 to 1/4

## Yaws

*(Framboesia)*

**Definition** A specific infective granuloma

**Etiology** Yaws is caused by the Treponema pertenue This cannot be distinguished microscopically from the causative organism of syphilis It is not a venereal disease and may be conveyed by direct contagion through a cutaneous abrasion and possibly by insect bites *Predisposing causes* 1 Locality East and West Africa the Malay States Philippine Islands Fiji and Samoa Burma Ceylon, Brazil and West Indies 2 Age It is not congenital and occurs especially in children and young people Over 60% of cases occur before the age of 15 years 3 Race Patients are usually natives

**Pathology** The ' Yaw is an infective granuloma The disease is distinct from syphilis but the Wassermann reaction is positive in the majority of cases

**Incubation Period** Probably a month or longer

**Clinical Findings** The onset is insidious with malaise headache pains in the muscles and bones and there may be slight fever The primary lesion may not be detected and may be single or multiple It generally occurs below the knees or near the mouth and is called the Mother Yaw It is a papule varying in size up to 2 or 3 inches in diameter which becomes nodular exudes fluid and forms a scab The secondary stage Some desquamation of skin occurs in about 3 months time followed by the appearance of more papules which protrude become red resembling a raspberry (*framboesia*) and crust over They may occur in any part of the body and after some weeks eventually drop off They are painless but may itch and treponemes are found in smears Tertiary lesions form from ulceration of the Yaw

*Varieties* 1 " *Crab* ' *yaws* affect the sole of the foot 2 ' *Gangosa* is an ulcerating yaw in the palate 3 *Goundou* is a nodular swelling on the nose 4 *Juxta articular nodules* may form tumours near the knees or elbows

**Differential Diagnosis** The disease can be distinguished from syphilis by the absence of a primary genital lesion and the absence of nervous manifestations such as tabes and general paralysis

**Course and Complications** The average duration if untreated is about a year but recurrences of secondary lesions may take place

**Prognosis** The disease is not usually fatal

**Treatment** Penicillin is the drug of choice The dose is an intra muscular injection of 1 2 million units of a procaine preparation in 2% aluminium monostearate for persons over the age of 10 years and 600 000 units for children under the age of 10 Two injections a week for 2 weeks are usually sufficient for a cure

## Dengue

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**Course and Complications** The course is usually as described Complications include Epistaxis bleeding from the gastro intestinal tract or uterus boils periarthritis of the knees or ankles and post dengue debility

**Prognosis** This is good The disease is practically never fatal

**Treatment** *Prophylactic* This is concerned with elimination of mosquitoes and prevention of their bites as for yellow fever

*Curative* The patient should be kept in bed until the rash has gone A saline aperient should be given at the onset Drugs are required for

there are two different varieties with very similar clinical features The organisms live in rats man is infected by their bite or by the bite of an infected ferret *Predisposing cause* Locality The disease occurs in Japan in parts of India such as Bombay and sporadically in other parts of the world

**Pathology** The lymph glands may be enlarged and hæmorrhages seen in the lungs The spleen is enlarged and the spirillum is found in the internal organs

**Incubation Period** This varies up to about 2 months

**Clinical Findings** The bite wound usually heals, but after a week or so breaks down with enlargement of the neighbouring lymphatic glands The patient now complains of headache, aching in the limbs and there may be a rigor with nausea or vomiting

*On Examination* There are periodical rises of temperature up to about 102° F lasting 2 to 5 days followed by a crisis and an afebrile interval of 1 to 2 days A purplish papular rash may be seen on the arms or trunk during the fever Conjunctivitis and small painful swellings in the muscles have been noted The relapses may continue for several months The blood The spirillum or streptobacillus may be found in the blood during pyrexial stages There is usually a leucocytosis of about 15 000 per c mm with some eosinophilia and a positive agglutin ation reaction to the spirillum The blood Wassermann reaction is positive

**Differential Diagnosis** The history of the bite and finding the organism in the blood differentiate rat bite fever from such diseases as relapsing fever and trench fever

**Course and Complications** The course is usually prolonged unless cut short by treatment Nephritis may occur

**Prognosis** The disease is serious unless adequately treated

**Treatment** Crystalline penicillin should be injected intramuscularly in doses of 30 000 units every 6 hours for 7 days for spirillum minus infections and for streptobacillus infection streptomycin is injected intramuscularly 1 G every 12 hours until the temperature has been normal for 2 days

## Leishmaniasis

A group of diseases due to infection with protozoa of the Leishman donovan type The group includes kala azar and tropical sore

## Kala azar

(*Dumdum Fever Black Fever*)

**Definition** A disease characterised by enlargement of the spleen, cachexia and irregular fever due to infection with a specific protozoon

**Etiology** The protozoon causing kala azar is the Leishman donovan body (Leishmania donovani) The protozoon appears in man as a small oval or cockle shaped body about 1 3 × 3μ with two nuclei Although the parasite develops readily in the sand fly *Phlebotomus argentipes* infection of man by the bite of this fly has not been proved

## Phlebotomus Fever

*(Sand fly Fever  Papataci Fever  Three day Fever)*

**Definition** *A specific disease* characterised by headache and generalised pains, due to the bite of a sand fly

**Etiology** Phlebotomus fever is caused by an ultra microscopic filterable virus which is present in the patient s blood during the first 1 or 2 days of the illness It is carried by a sand fly (*Phlebotomus papatasi*) The virus probably undergoes changes in the fly s body, as the latter does not become infective for 6 days after biting a patient *Predisposing causes* 1 Locality The disease occurs in the eastern part of the Mediterranean, and in Mesopotamia India and Persia 2 Climate Warmth and moisture are necessary

**Pathology** There are no post mortem findings

**Incubation Period** This is 4 to 7 days

**Clinical Findings** The bite of the fly produces some local irritation and swelling There may be malaise for a day or so before the onset, which is usually sudden with a rigor The patient complains of frontal headache, pains behind the eyes in the neck, back and limbs There is often insomnia

*On Examination* The face is flushed and the conjunctivæ are red The throat is congested and some vesicles may be seen on the mucous membrane The tongue is furred in the centre The temperature rises rapidly to 103° or 105° F but the pulse remains slow, about 70 or 80 The blood There is usually a leucopenia The temperature falls in about 2 days and there may be some diarrhœa, epistaxis and sweating The pulse becomes slower, 40 to 50 There is no rash

**Differential Diagnosis** Sand fly fever must be distinguished from malaria influenza and dengue Examination of a blood film excludes malaria , the course of the disease and its occurrence where there are sand flies usually serve to establish a diagnosis

**Course and Complications** The usual course is as described above Rarely a recrudescence occurs with a rise of temperature to a lesser degree for a short time about the fifth or sixth day

**Prognosis** This is good The disease is never fatal

**Treatment** *Prophylactic* Sand flies should be destroyed and their breeding places eliminated by spraying with D D T Dimethyl phthalate should be applied to the wrists and ankles at night Mosquito nets should be sprayed with D D T

*Curative* Liq iodi mitis should be applied to the bites The patient should be kept in bed for 5 or 6 days and the pains relieved by aspirin gr 10 t d s

## *Rat bite Fever*

*(Sodoku)*

**Definition** A disease caused usually by the bite of a rat, infected with a special spirillum

**Etiology** Rat bite fever is caused by the Spirillum minus (Spirochæta morsus muris) or by the Streptobacillus *moniliformis* as

*Curative* If there is severe anæmia a blood transfusion should be given Pentavalent antimony preparations such as Pentostam are very efficient Fourteen daily intravenous injections are given of 0 3 G For cases which are resistant to antimony especially those occurring in the Sudan the aromatic diamidines are available, such as stilbamidine and pentamidine isethionate The latter is given daily intramuscularly or intravenously for 12 to 15 days the usual dose being 2 mg /kg body weight Headache giddiness flushing abdominal pain or low blood pressure may be relieved by the oral administration of Anthisan mg 100 half an hour before the injection

## Tropical Sore

*(Dermal Leishmaniasis Delhi Boil Oriental Sore Baghdad Boil Aleppo Sore)*

The causative organism is the Leishmania tropica which cannot be distinguished from the L donovani Ulceration occurs in the skin and on mucous membranes the disease is probably spread by a sand fly, the *Phlebotomus papatasi* In the American tropics this form of leishmaniasis is called Espundia

Local treatment consists in the application of solid carbon dioxide pure phenol superficial X rays or radium In addition 2 ml of a 5% solution of emetine hydrochloride may be injected into the periphery of the lesion Other substances used for local infiltration include berberine sulphate (2%) and Neostam (2%)

Infantile kala azar is due to the Leishmania infantum which is probably the same protozoon *Predisposing causes* 1 Locality Kala azar was first described in Assam, it occurs in other parts of India, in China, the Sudan, West Africa, etc Infantile kala azar is found around the Mediterranean It is more prevalent in the country than in towns 2 Climate Warmth and moisture favour its appearance

**Pathology** The Leishman donovan body occurs in endothelial cells, especially in the spleen in the blood it may be present in polymorphonuclear leucocytes A flagellate stage is found in certain sand flies and can be obtained on culture of the cockle shaped bodies Post mortem, the spleen is enlarged and somewhat firm the liver is also a little enlarged and ulcers may be found in the small and large intestine The marrow of the long bones is unduly red

**Clinical Findings** The incubation period is uncertain, varying from a few months to over a year The disease usually begins insidiously, with progressive weakness loss of weight and malaise there may be diarrhœa, sweating or bleeding from the nose or gums In some cases the onset is more sudden suggesting malaria and in others resembling typhoid fever

*On Examination* In an established case the patient is wasted and in Europeans the skin is seen to be pigmented The spleen is enlarged and the liver may be palpable The temperature is irregularly raised and may present 2 or 3 summits in the 24 hours The pulse is proportionately frequent The blood There is anæmia and leucopenia is pronounced about 2 000 per c mm Leishman donovan bodies are seen at times in polymorphonuclear leucocytes There is an excess of gamma globulin in the blood Diagnosis may be confirmed by withdrawing some splenic tissue by spleen puncture making a film and staining for Leishman donovan bodies Sternal puncture will also show the Leishman donovan bodies in the monocytes and this is a safer method of diagnosis than is splenic puncture

**Differential Diagnosis** Other causes of chronic enlargement of the spleen (see p 538) should be excluded if there is doubt as to the diagnosis A blood examination usually excludes malaria as does the response to anti malarial treatment Finding the protozoon by sternal or spleen puncture settles the diagnosis

**Course and Complications** The average course of the disease if untreated, is from a few months to 2 years when death occurs from progressive weakness or some septic complication, such as pneumonia or gangrene of the lungs

**Prognosis** The mortality has been lowered by antimony treatment from a previous 96% to about 2% except in the Sudan where very severe and drug resistant cases are met with

**Treatment** *Prophylactic* Healthy natives in a kala azar district should be kept together at night and away from those infected with the disease The sand fly range is only a few hundred yards D D T sprays should be used and dimethyl phthalate smeared on the skin and clothing Nets to afford protection would have to be of too fine a mesh to be tolerated at night

**Locality** World wide distribution

**Hosts** Definitive Man Intermediate The pig or rarely man (*Somatic tæniasis*)

**Length of Adult Worm** About 10 feet

**Head.** 1 mm in diameter Globular with 4 suckers and a rostellum armed with 2 rows of 14 hooklets Neck thin

**Segments** About 1 000 Contain a uterus with about 10 lateral branches the genital pore is lateral and alternates regularly Mature segments are three times as long as they are broad (see Fig 68)

**The Ova** Circular Diameter about 35 $\mu$

**The Onchosphere (embryo)** About 20 $\mu$ in diameter Has 6 hooks

**The Cysticercus Cellulosæ** Like a little bladder oval about 5 to 20 mm long Single scolex forms in it

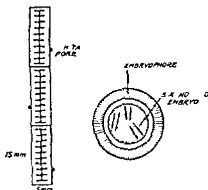



Fig 68 T Solium Mature Segments (Natural size )

T solium fæcal ovum (onchosphere) brown circular 35 $\mu$ in diameter (× 500 )

Segments containing ova are passed in man s fæces and are eaten by the pig Rarely *somatic tæniasis* occurs in man either by transference of ova by his fingers to the mouth or owing to segments containing ova being regurgitated into his stomach The cysticerci (bladder worms) may then develop under the skin as small nodules or in the brain causing epilepsy in the eyes, or muscles They may be revealed by X ray examination and must not be confused with the cysts of the Trichinella spiralis The latter are probably too small to be seen radiologically

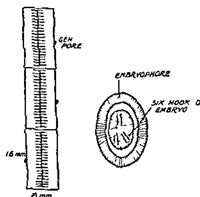



Fig 69 T Saginata Mature Segments (Natural size )

T saginata fæcal ovum (onchosphere) brown oval 38 $\mu$ × 25 $\mu$ (× 500 )

### The Tænia Saginata

(*Tænia Mediocanellata The Unarmed Tape Worm The Beef Tape Worm The Fat Tape Worm*)

**Infection** By eating imperfectly cooked beef infected with the Cysticercus bovis

**Locality** World wide distribution

**Hosts** Definitive Man Intermediate Cattle

***Length of Adult Worm*** *About 20 feet*

***Head*** 2 mm in diameter Pear shaped with 4 suckers but no rostellum and no hooklets Neck medium size

**Segments** About 2 000 Contain a uterus with about 20 lateral

# CHAPTER XIV

# THE PARASITIC WORMS

## THE CESTODES

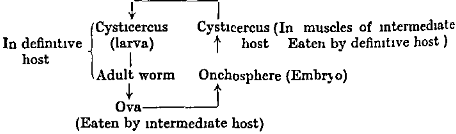
### (*Tape Worms*)

**Definition** Segmented flat worms usually hermaphrodites

**Anatomy** The tape worms consist of a head and neck (scolex) and segments (proglottides) The segments near the head are immature, the intermediate ones contain sexual glands and the terminal ones ova They have no alimentary canal

*Varieties* The most important varieties are the Tænia solium, the Tænia saginata, the Diphyllobothrium latum (Dibothriocephalus latus), the Echinococcus granulosus (Tænia echinococcus) and the Hymenolepis nana (Tænia nana) Man is infected by eating the larvæ in meat or fish which is raw or insufficiently cooked The larva is known as a cysticercus if it forms one cyst and one scolex (as in T solium and T saginata) and as an echinococcus if it forms many cysts and many scolices in each cyst (as in E granulosus) The larva of the D latum is a plerocercoid and elongated like a worm The larva develops in the definitive host into the adult worm, and the eggs which pass out in the fæces are eaten by the intermediate host The outer layer is dissolved in the alimentary canal and the contained embryo (onchosphere) is liberated This passes into the tissues to form the larva (cysticercus)

*Cycle*




The ovum of the D latum forms a ciliated embryo this is eaten by certain crustacea (such as *cyclops strenuus*) and forms a procercoid larva The cyclops containing the procercoid is eaten by a fish and becomes encysted as a plerocercoid larva Man eats the fish and is infected the larva developing into the adult worm In E granulosus infection man eats the ova and the larva formed develops in his tissues the adult form occurring in animals such as the dog

### The Tænia Solium

(*The Solitary Tape Worm The Armed Tape Worm The Pork Tape Worm*)

The chief features are as follows

**Infection.** By eating imperfectly cooked measly ' pork containing the larvæ (Cysticerci cellulosæ)

**Locality** World wide distribution

**Hosts** Definitive Man Intermediate The pig or rarely man (*Somatic tæniasis*)

**Length of Adult Worm** About 10 feet

**Head.** 1 mm in diameter Globular with 4 suckers and a rostellum armed with 2 rows of 14 hooklets Neck thin

**Segments** About 1 000 Contain a uterus with about 10 lateral branches the genital pore is lateral and alternates regularly Mature segments are three times as long as they are broad (see Fig 68)

**The Ova** Circular Diameter about 35 $\mu$

**The Onchosphere (embryo)** About 20 $\mu$ in diameter Has 6 hooks

**The Cysticercus Cellulosæ** Like a little bladder oval about 5 to 20 mm long Single scolex forms in it

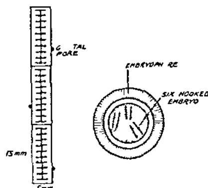



Fig 68 T Solium Mature Segments (Natural size)

T solium fæcal ovum (onchosphere) brown circular 35 $\mu$ in diameter (× 500)

Segments containing ova are passed in man s fæces and are eaten by the pig Rarely *somatic tæniasis* occurs in man either by transference of ova by his fingers to the mouth or owing to segments containing ova being regurgitated into his stomach The cysticerci (bladder worms) may then develop under the skin as small nodules or in the brain causing epilepsy in the eyes or muscles They may be revealed by X ray examination and must not be confused with the cysts of the Trichinella spiralis The latter are probably too small to be seen radiologically

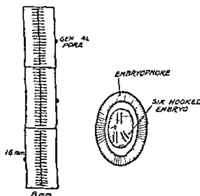



Fig 69 T Saginata Mature Segments (Natural size)

T saginata fæcal ovum (onchosphere) brown oval 38 $\mu$ × 25 $\mu$. (× 500)

### The Tænia Saginata

(*Tænia Mediocanellata The Unarmed Tape Worm The Beef Tape Worm The Fat Tape Worm*)

**Infection** By eating imperfectly cooked beef infected with the Cysticercus bovis

**Locality** World wide distribution

**Hosts** Definitive Man Intermediate Cattle

**Length of Adult Worm** About 20 feet

**Head** 2 mm in diameter Pear shaped with 4 suckers but no rostellum and no hooklets Neck medium size

**Segments** About 2 000 Contain a uterus with about 20 lateral

branches the genital pore is lateral and alternates irregularly, the mature segments are twice as long as they are broad (see Fig 69)

**The Ova** Oval measuring 38 μ by 25 μ approximately

**The Onchosphere** (embryo) Head has 6 hooks

**The Cysticercus Bovis** 8 mm by 5 mm Single scolex forms in it

The segments containing ova are passed in man s fæces and are eaten by the ox

### The Diphyllobothrium Latum

(*The Dibothriocephalus Latus The Russian Tape Worm*)

**Infection** By eating caviare or imperfectly cooked fish

**Locality** Russia the Baltic Switzerland

**Hosts** Definitive Man Intermediate Fish such as pike perch salmon trout and grayling

**Length of Adult Worm** About 30 feet

**Head** 3 mm long Olive shaped with a suction groove on its dorsal and ventral sides There are neither rostellum nor hooklets

**Segments** About 3 000 Contain a central rosette uterus and a ventral genital pore They are broader than they are long (see Fig 70)

**The Ova** Oval measuring about 60 μ by 40 μ with an operculum

**The Onchosphere** Ciliated and free swimming It is eaten by crustacea (such as the *Cyclops strenuus*)

**The Procercoid Larva** Elongated oval 5 μ long

**The Plerocercoid Larva** Worm like about 6 mm long Man passes free ova in his fæces

**Clinical Findings** Infestation of man with T solium T saginata, or with D latum may give rise to vague symptoms of indigestion or to marked hunger with bodily wasting A severe anæmia of the pernicious type rarely occurs with D latum infestation It is estimated that in Finland 250 000 people are infested but only 0 5% suffer from anæmia





Fig 70 The Diphyllobothrium Latum Mature Segments (Natural size)

Diphyllobothrium latum fæcal ovum brown oval 60 μ × 40 μ (× 250)

**Treatment** *Prophylactic* Meat and fish must be adequately inspected for cysticerci, and properly cooked

*Curative* The patient is kept in bed and starved for 2 days During this time only fluids are allowed such as fruit juices with glucose tea black coffee and meat extracts An initial dose of castor oil fl oz ½ is given and this is followed by mag sulph gr 60 t d s on the second day On the third day no food is allowed until the worm is passed and the patient is kept recumbent Mag sulph gr 240 is administered at 7 a m and after the bowels have acted ext filicis liq m 15 in a capsule or emulsion is given at 9 a m at 9 15 a m at 9 30 a m and at 9 45 a m and m 30 at 10 a m followed at noon by mag sulph gr 240 Alternatively mepacrine can be given instead of filix mas The dose for an adult is 1 G (ten 0 1 G tablets) in a glass of water to which is added two teaspoonfuls of sodium bicarbonate Sod sulph gr 240 must be given 1 hour before and 2 hours after the

*Hydatid of the Brain* This causes symptoms of a cerebral tumour
*Hydatid of the Heart* This may cause sudden death

The blood may show an eosinophilia and the Casoni intradermic test is positive (see p 84)

**Course and Complications** The cyst may grow to 5 or 6 inches in diameter, it may atrophy calcify or its contents may suppurate Rupture may occur into the peritoneum, the stomach intestines the pleura, the inferior vena cava or the bile ducts Rupture may be accompanied by shock and by urticaria, and in cases of intraperitoneal rupture secondary cysts may form

**Prognosis** The disease is serious Frequently, however the cyst dies and inspissates Suppuration or rupture make the outlook more grave

**Treatment** There is no medical treatment If possible, the cyst should be removed surgically without rupture

### The Hymenolepis Nana

(*The Tænia Nana The Dwarf Tape Worm*)

This worm infests especially children in America Egypt and Southern Europe It is 25 to 40 mm long and 1 mm broad It has a rostellum with 4 suckers and a single row of about 30 hooklets There are about 200 segments There is probably no intermediary host the eggs which are liberated in the intestine developing into adult worms

**Treatment** Ol chenopodii may be given The dose for children is one drop for each year of age on a lump of sugar followed 2 hours later by a dose of castor oil appropriate to the age of the child Alternatively a dose of sodium sulphate is given the night before, and next morning while fasting, a hard gelatin capsule containing 0 4 G Caprokol is taken Two hours later a second dose of Glauber's salts is given For children over 5 years the dose of Caprokol is 0 6 to 0 8 G

## THE TREMATODES

(*Flukes*)

The trematodes are flat non segmented leaf like worms usually hermaphrodites possessing a mouth and 1 or more suckers The most important varieties which are parasitic in man are the schistosoma and the paragonimus (lung fluke) the fasciola or liver fluke very rarely infests man

### Schistosomiasis

(*Bilharziasis*)

There are three varieties

### Urinary Schistosomiasis

(*Endemic Hæmaturia*)

**Etiology** Endemic hæmaturia is caused by the Schistosoma hæmatobium (Bilharzia hæmatobium) Man is infected by cercariæ

(young swimming forms of the trematode) which pierce his skin when bathing or paddling and infection may occur by drinking water The cercariæ pass to the portal vein and in about 6 weeks develop into the adult male and female forms of the S hæmatobium The male is flat about 15 mm by 1 mm and the female is slender and round about 25 mm (1 inch) by 0 25 mm They migrate against the blood stream from the portal to the pelvic veins and veins of the bladder or rectum, the male curves from side to side to form a gynæcophoric canal in which he embraces the female when they have reached the small veins the female leaves the male and migrates to the smallest vein allowing her passage The ova are then extruded The ovum is about 190 $\mu$ by 70 $\mu$ and has a sharp terminal spine, this penetrates the blood vessel and the ovum is pushed through the vessel by the blood stream It then ulcerates its way into the submucous tissues and through the mucous membrane of the bladder or rectum The ova are chiefly passed in the urine On coming in contact with water an embryo or ciliated miracidium hatches out This enters a spiral fresh water snail, such as the *Bullinus contortus* Sporocysts form and cercariæ are liberated into the water from the snail, completing the cycle The disease is met with in Africa Arabia and Iraq

**Pathology** Post mortem the mucous membrane of the bladder is red thickened in patches and polypi may be seen The muscular wall is hypertrophied The prostate may be enlarged and the ureters dilated Ova may be seen in the submucous tissues of the bladder or in the prostate the liver may be cirrhosed

**Incubation Period.** 1 to 3 months

**Clinical Findings** The first symptoms appear a few weeks after infection They constitute the toxæmic stage The patient complains of urticaria malaise with perhaps abdominal pain

*On Examination* The temperature is raised and the blood shows an eosinophilia which may reach 50% A positive complement fixation may be obtained by using as an antigen an extract of the liver of infected snails

Local symptoms may not be noticed for a few months or even years The patient then complains of passing blood usually at the end of micturition with perhaps frequency scalding and pain in the perineum or suprapubic region He may also pass blood and mucus in his motions

*On Examination* The patient is often pale or sallow The urine The deposit shows ova red corpuscles and pus cells The blood may show a leucocytosis of about 15 000 per c mm with an eosinophilia of about 12% The complement fixation test is usually still positive Cystoscopy may reveal an inflamed vesical mucous membrane and white patches may be seen due to submucous ova

**Differential Diagnosis** Other causes of hæmaturia such as a calculus, cystitis etc must be excluded The presence of ova in the urine is diagnostic but a calculus may also be present

**Course and Complications** The disease, if untreated pursues a chronic course for many years and if the patient is not destroyed by

complications, spontaneous cure usually results Complications include Cystitis, vesical calculus, hydronephrosis, pyonephrosis, carcinoma of the bladder urethral fistula or stricture, etc

**Prognosis** This depends upon the severity of infection, the presence of complications and the institution of adequate treatment

**Treatment** *Prophylactic* Bathing or paddling in endemic areas should be forbidden and drinking water should be boiled The snails may be destroyed by cutting off the water of the irrigation canals periodically for 2 weeks during the dry season Carriers should have their infestation cured by treatment

*Curative* Sodium antimony tartrate is injected slowly into a vein The injection should be given about 2 hours after a meal A 0 5% solution is used on the first day 8 ml on the third day 12 ml on the fifth day 16 ml on the seventh day 20 ml and on the ninth day and on subsequent alternate days 24 ml until a total of 320 ml has been injected The hæmaturia may be temporarily increased The urinary deposit should be examined daily for ova which, as they are killed shrivel, darken and when placed in water will not hatch out A cough is often noticed during the injection This is due to the antimony reaching the lungs If toxic symptoms follow an injection the next dose should not be increased Toxic symptoms include tightness of the throat or chest abdominal pain and a metallic taste If there is collapse at the injection Pituitrin 1 ml or liq adrenal hydro chlor 0 5 ml should be injected intramuscularly If the patient has heart or kidney disease the antimony is contraindicated When the veins are difficult stibophen (Fouadin) also a trivalent antimony preparation may be injected intramuscularly using a 6 3% solution Ten to fifteen injections are given on alternate days For an adult the dose is 1 5 ml followed by 3 ml the subsequent doses being 5 ml Good results have also been obtained in urinary schistosomiasis with Nilodin (miracil D) It is put up in tablets of mg 200 and administered by mouth The dose is 22 mg /kg body weight for 16 days

## Intestinal Schistosomiasis

**Etiology** This disease is caused by the Schistosoma mansoni (Bilharzia mansoni) The adult trematodes are found in the mesenteric veins and their ova pass through into the rectum The ova have a lateral sharp spine The intermediate host is a flat fresh water snail the *Planorbis boissyi* The life history resembles that of the S hæmatobium Intestinal schistosomiasis occurs in Egypt the Belgian Congo Nigeria, the West Indies and the northern part of South America

**Pathology** Post mortem the mucous membrane of the rectum and colon is thickened, and there are papillomatous swellings The liver may be fibrotic ( 'pipe stem' cirrhosis) and ova are found in it

**Clinical Findings** An early toxæmic stage with fever and urticaria occurs followed in about 2 months time by dysenteric symptoms, with blood and mucus in the motions The ova are found in the fæces, and the blood shows an eosinophilia and gives a positive complement fixation test In Egypt a special type occurs associated with enlarge

ment of the liver and spleen Nodular lesions may form in the lungs which may lead to pulmonary endarteritis and right heart failure The brain may also be involved with hemiplegia or epileptiform convulsions Anæmia and ascites are present This variety is called Egyptian splenomegaly

**Treatment** This is by sod antimon tartrate or stibophen as for urinary schistosomiasis (see above)

### Eastern Schistosomiasis

**Etiology** Eastern schistosomiasis is caused by the Schistosoma japonicum (Bilharzia japonica) The trematodes are found in the mesenteric veins and the ova pass into the large intestine The ova have no spines but a small lateral knob may be seen The intermediate host is the *Hemibia japonica* a fresh water mollusc The life history resembles that of the types described above The disease is met with in China South Japan the Philippine Islands and Upper Burma

**Pathology** Post mortem the liver is enlarged cirrhotic and contains ova the spleen is enlarged but usually free from ova ova may be found in the brain The mucous membrane of the large intestine is thickened and polypi may be seen

**Clinical Findings** An early toxæmic stage occurs with fever urticaria and eosinophilia Later there are abdominal symptoms with pain and dysentery

*On Examination* The liver and spleen are enlarged and there may be ascites The ova are found in the fæces

**Treatment** This consists in giving injections of sodium antimony tartrate or stibophen as described above

### Paragonomiasis

**Etiology** Paragonomiasis is caused by the Paragonimus westermani (Distoma ringeri) The fluke is about $\frac{1}{3}$ inch (8 mm ) long

**Pathology** The parasites occur in the lungs

**Clinical Findings** The flukes are the cause of endemic hæmoptysis

### Fascioliasis

**Etiology** Fascioliasis is caused by the *Fasciola hepatica* (*Distoma hepaticum*)

**Pathology** The flukes are found chiefly in the livers of sheep Man is infected by eating raw water cress

## THE NEMATODES

These are cylindrical non segmented unisexual worms The following are the most important varieties occurring in man Ascaris lumbricoides Enterobius (oxyuris) vermicularis Trichuris trichiura (trichocephalus dispar) Trichinella spiralis Strongyloides stercoralis Ankylostoma duodenale Necator americanus Wucheria (filaria) bancrofti Loa loa Dracunculus medinensis and the Onchocerca volvulus

## Ascariasis

### (*Round Worm Disease*)

**Etiology** Ascariasis is caused by the Ascaris lumbricoides

*The Adult Worm* Male, average size 20 cm (8 inches) in length and 0 3 cm ($\frac{1}{8}$ inch) in diameter Female, average size 30 cm (12 inches) in length and 0 5 cm ($\frac{1}{5}$ inch) in diameter They are light brown in colour with pointed ends, and live in the small intestine in man and in animals such as the pig Their eggs, which measure about 70 $\mu$ by 60 $\mu$, are passed in the fæces Man is infected by swallowing the eggs in water or contaminated uncooked food stuffs When ingested, the larvæ which have developed inside the eggs, pass out and are believed to make their way to the liver and lungs, then to the trachea larynx, œsophagus stomach and intestine, where they develop into adult worms Infestation is more common in children than in adults

**Pathology** Post mortem the worms are found chiefly in the upper part of the small intestine and they may cause obstruction of the bile or pancreatic ducts, of the appendix or of the intestine They may also perforate the intestine and cause peritonitis

**Clinical Findings** The patient may be unaware of their presence, or complain of vague symptoms of indigestion flatulence cutaneous irritation or urticaria Fretfulness and convulsions occur in children In some cases asthma or dysenteric symptoms are present The worms may enter the stomach and be vomited or pass up the œsophagus during the night coming out through the mouth or nose When the larvæ are traversing the lungs they may give rise to a febrile disturbance with cough and expectoration On examination of the fæces the characteristic ova are found or a worm is seen

**Course and Complications** The patient often remains infected for several years, but the worms tend to die unless auto infection from the ova in the patient's own fæces occurs Such complications as appendicitis, intestinal obstruction or perforation, jaundice, pancreatitis or laryngeal obstruction may rarely occur

**Treatment** *Prophylactic* Uncooked vegetables should be well washed before consumption

*Curative* Caprokol (hexyl resorcinol B P C ) is more efficacious than santonin but care is required in its use Sod sulphate gr 240 to 480 should be given in water the night before Caprokol is given on an empty stomach and no food must be taken for 4 hours, when a second dose of sod sulphate is given Caprokol is put up in hard gelatin capsules each containing 0 15 G These must not be chewed The dose is 0 16 to 0 85 G for a child and 1 G for an adult The stools should be subsequently examined for ova If they are present the treatment must be repeated to expel the remaining worms Satisfactory results are also obtained with Antepar Elixir This contains as its active principle, piperazine citrate equivalent to mg 500 piperazine hydrate in m 60, in stable combination The dose is 50 to 75 mg /kg body weight for 7 days

## Enterobiasis

(*Oxyuriasis Threadworm Disease Pinworm Disease*)

**Etiology** The disease is caused by the Enterobius (oxyuris) vermicularis

*The Adult Worm* The male average length 3 mm The female average length 10 mm The worms are white and threadlike and live in the cæcum and colon The female passes outside the anus and lays her eggs The patient scratches and the ova are transferred on the fingers to the mouth the ova hatching out in the intestine The eggs measure about 50 μ by 25 μ and are flat on one side Man is infected by ingesting the ova in water or by contaminated uncooked food stuffs Air borne infection may also occur as ova are present on objects at all levels in all rooms in infested households familial infestation being very common Auto re infection is very liable to occur Fertilisation takes place in the small intestine after which the male worm dies and the female passes to the cæcum as described above The disease occurs in adults and in children

**Clinical Findings** Threadworms are an important cause of restlessness and irritability in children In adults they should be remembered as a cause of pruritus ani Children complain of itching of the anus and there may be frequency of micturition vaginitis or prolapse of the rectum Ova are not often found in stool examination and the best method of detecting infestation is by using a swab consisting of a glass rod tipped with a small square of cellophane This is stroked over the perianal region and the cellophane is placed between a glass slide and a cover slip and examined for ova

**Treatment** *Prophylactic* Uncooked vegetables should be well washed before consumption

*Curative* All infested members of a household must be treated The fæces should be examined for ascaris ova and if present the ascaris infestation should first be treated The hands should be scrubbed after the bowels are opened before meals before the morning cup of tea and after taking off any article of clothing in contact with the skin The nails must be kept short and a child should wear gloves and tight fitting knickers at night to prevent scratching Antepar Elixir should be given as described on p 754

## Trichuriasis

(*Whipworm Disease*)

**Etiology** Trichuriasis is caused by the Trichuris trichiura (trichocephalus dispar)

*The Adult Worm* The male average length 40 mm (1½ inches) The female average length 50 mm (2 inches) The worms are grey in colour the anterior extremity ends in a fine thread The worms are found in the cæcum and colon The eggs are brown barrel shaped with an albuminous plug at each end and measure about 50 μ by 22 μ Man is infected by water and contaminated uncooked food The larva

is liberated from the ovum in the stomach and probably passes through the liver, lungs, trachea and œsophagus to the intestines The worms are said to pierce the mucous membrane with their fine extremity which prevents their being dispelled by anthelmintics

**Clinical Findings** The worms usually produce no symptoms, but may cause appendicitis or chronic diarrhœa with a microcytic hypochromic anæmia and eosinophilia The ova are found in the fæces

**Treatment** Ol chenopodii may be given The dose for children is 1 drop for each year of age on a lump of sugar followed 2 hours later by sod sulph gr 120 to 240 Alternatively hexyl resorcinol may be given, 1 G by mouth in a hard gelatin capsule first thing in the morning No food is taken for 6 hours and next morning the bowels are opened with mist mag sulph alba (B P C) fl oz 1

## Trichiniasis

(*Trichinosis Trichinelliasis*)

**Etiology** Trichiniasis is caused by the Trichinella spiralis

*The Adult Worm* The male average length 1 6 mm diameter 0 04 mm The female average length 4 mm, diameter 0 06 mm Man is usually infected by eating raw or partially cooked ham, pork or pork sausages containing encysted trichinellæ Pigs are infected either by eating raw garbage containing pieces of infected pork or by eating infected rats The former method is very prevalent in America Larvæ develop from the cysts and form adult worms which live in the small intestine The larvæ produce embryos which pass by lymphatics or blood vessels to striated muscles and encyst coiled up near the tendinous insertions of the muscles They are found especially in the intercostal muscles diaphragm, the muscles of the neck, upper arm and calf Calcification may occur in the cyst wall The disease is common in America and in Germany

**Incubation Period** This varies from 5 to 16 days

**Clinical Findings** Three stages were originally described *The Invasion Stage* This is characterised by malaise vomiting and watery diarrhœa with fever and at times delirium *The Migration Stage* During the second week of the illness pains occur in the arms and legs There may be difficulty in breathing or in mastication An urticarial rash may also appear with œdema of the legs and face The blood shows a leucocytosis of about 20 000 per c mm with an eosinophilia of about 40% Adult worms are rarely found in the fæces *The Encystment Stage* This usually gives rise to no symptoms

Epidemics occurred in England in 1941 in which the preliminary gastro intestinal symptoms were very slight and in some cases con stipation was a prominent feature The onset was characterised by fever of 101° to 102°F swelling of the eyelids face and occasionally limbs, and frontal headache 'Splinter hæmorrhages were seen under the nails In some cases meningeal or encephalitic symptoms were present and in about half the cases a dry cough was an initial

symptom Muscular pains were noted about 3 days after the onset, and temporary mental changes such as melancholia, were not infrequent The urine was usually free from albumin and the blood showed an eosinophilia of about 40% Asymptomatic infestation also occurs in which no symptoms of the disease are manifested during life but post mortem encysted worms are found in the diaphragm

**Differential Diagnosis** Trichiniasis may be mistaken for acute nephritis or sinusitis owing to the orbital œdema The muscular pains may suggest acute rheumatism The nervous symptoms may lead to a diagnosis of meningitis or of encephalitis The fever and gastro intestinal symptoms may resemble those of food poisoning or enterica group infections The initial fever and cough may cause confusion with influenza or bronchitis The correct diagnosis is suggested by the fever leucocytosis and eosinophilia It is established by finding the larvæ in the blood during the first 2 or 3 weeks of the illness For this purpose 5 ml of blood are laked with 10 ml of water and the centri fugalised deposit is examined microscopically The adult worms are seldom found in the fæces Muscle biopsy will demonstrate the presence of T spiralis in the majority of cases after the fourth week of the illness a small portion of the pectoralis major deltoid or gastrocnemius muscle is examined Intradermic injection of a saline suspension of powdered larvæ gives a positive reaction as shown by the formation within 5 minutes of a wheal and an erythematous zone in nearly all people for 6 months after infestation Reports that calcified cysts can be demonstrated radiologically in the muscles in long standing cases are probably incorrect The cysts are usually too small to be thus revealed and confusion has arisen by mistaking encysted cysticerci for them

**Course and Complications** The feverish stage may last for 2 to 3 weeks and the muscular pains and weakness for several months Death may occur from myocarditis or encephalitis

**Prognosis** In an outbreak in Berlin in 1930 10 out of 70 cases proved fatal The mortality was 16% in a series of 1 550 cases in America and no deaths occurred in 500 cases in the Wolverhampton neighbourhood in the English epidemic of 1941

**Treatment** *Prophylactic* Infected meat must be avoided and ham, pork and pork sausages should be well cooked

*Curative* During the invasion period the patient should be given calomel gr 3 followed by mag sulph gr 120 in the morning or castor oil fl oz ½ to drive out the worms before the embryos are formed This treatment should be repeated again the next day There are no measures available for eliminating the cysts from the muscles

## Strongyliasis

The Strongyloides stercoralis lives in the submucous tissues of the small intestine in man The female worm measures 2 5 mm by 0 3 mm The male is not found in the intestine It is thought that the worms cause diarrhœa at times (Cochin China diarrhœa)

## Ankylostomiasis

(*Uncinariasis Hookworm Disease Miner's Anæmia*)

**Etiology** Ankylostomiasis is caused by a small nematode, of which there are two types, the Ankylostoma duodenale and the Necator americanus

The A duodenale The male is about 9 mm by 0 5 mm in size and the female slightly larger Its name implies that it has a bent mouth which contains 4 teeth It lives chiefly in the jejunum attached by its mouth to the mucous membrane from which it sucks blood Many eosinophil cells congregate at the site of its attachment

The N americanus This is slightly smaller Thousands of eggs are formed which are excreted from the human intestine When these come in contact with damp soil or water, embryos hatch out and enter the skin of man usually through the feet, producing ground itch They then pass in the circulation to the heart and lungs They burrow into the bronchi and are carried with the mucus up the trachea and pass down the œsophagus into the small intestine Locality The disease occurs throughout the sub tropical world, that due to the Ankylostoma duodenale being met with in the Mediterranean and Southern European areas also in certain districts in India and Northern China It has also been found amongst tin miners in Cornwall coal miners in Belgium and the workers in the St Gothard tunnel It is a disease of the country rather than of towns and affects children and adults The N americanus is found in North and South America and also in Central Africa India the Philippines Ceylon etc

**Pathology** Post mortem the body is well nourished but pale The heart liver and kidneys are fatty, and localised hæmorrhages are seen in the mucous membrane of the small intestine Serous effusions may be present Over a thousand worms may be found in the intestine

**Clinical Findings** The local lesions in the feet ('ground itch') result in the formation of vesicles and pustules which heal in a week or so It is several months before the general symptoms are felt The patient complains of progressive weakness shortness of breath and palpitations There may also be flatulence and constipation or diarrhœa The appetite is good but pica (dirt eating) is a feature of the disease

*On Examination* The complexion is pale or sallow The temperature may be slightly raised There may be œdema of the feet with dilatation of the heart The spleen is not enlarged The blood The anæmia is of a microcytic hypochromic type The characteristic feature is an eosinophilia of about 20% The stools usually show blood either by naked eye or by occult blood tests Ova are seen microscopically

**Differential Diagnosis** Other causes of anæmia are excluded by the eosinophilia and the presence of the ova in the fæces

**Course and Complications** The disease pursues a prolonged course if untreated and results in much economic loss amongst workers in the

tropics Growth is much diminished in infected children Dysentery with blood and mucus in the motions, polyarthritis or nephritis may occur

**Prognosis** This is usually good but at times the disease is rapidly fatal

**Treatment** *Prophylactic* Latrines should be provided and their usage enforced in endemic zones and the feet protected by boots Drinking water should be boiled

*Curative* Various anthelmintic drugs are employed Tetrachlor ethylene is of value It is put up in hard gelatin capsules containing 1 ml The dose is 3 ml for an adult and for a child 0 2 ml for each year of age up to 15 years in a single dose Caprokol is safer for small children and for debilitated adults The method of administration is described on p 754

Oil of chenopodium is also used No purge is given before the treat ment In the morning ol chenopodii m 8 is given in a hard gelatin capsule every hour for 3 doses Two hours later mag sulph gr 240 is given

Carbon tetrachloride is dangerous and should not be used as if impure it may produce toxic symptoms resembling delayed chloroform poisoning due to degeneration of the liver

## Filariasis

**Etiology** There are several types of these small nematodes which produce disease in man the most important being the Wucheria (filaria) bancrofti the Loa loa and the Onchocerca volvulus

### Wucheria bancrofti

(*Filaria bancrofti*)

Infection is conveyed to man by mosquito bites chiefly by the *Culex fatigans* The female mosquito introduces the embryos into human beings and they pass to the lymphatic vessels and glands, where they develop into the adult filaria The male is about 40 mm (1½ inches) long and the female twice its length They are very fine and resemble a coiled hair The female produces large numbers of embryos (microfilariæ) which pass into the blood stream They measure about 0 3 mm long by 7 5 $\mu$ wide These are taken up by a biting mosquito and after undergoing changes in its body are injected again into man The embryos are practically absent from the peripheral circulation of man during the day being located then chiefly in the lungs and kidneys They pass into the peripheral circulation *during* the night beginning to migrate before the patient goes to sleep reaching their maximum about midnight and diminishing in numbers before he awakes This corresponds with the night activity of the mosquito If the patient sleeps by day the periodicity is altered In the Pacific Islands the W bancrofti shows no periodicity, the intermediary mosquito the *Aedes variegatus* there biting during the day The disease occurs in the tropical portions of North and South America the West Indies

in North Africa Central Africa, China Japan, North Australia India and the Malay Archipelago

**Pathology** The changes produced by the adult filariæ result from lymphatic obstruction and secondary inflammation of lymphatics In tropical elephantiasis adult filariæ can often be found in the neighbouring lymphatic glands The lymphatic glands and vessels become fibrosed and the embryos cannot pass through them into the circulation The embryos are not known to produce any pathogenic effects

**Incubation Period** This is probably a matter of 4 or 5 years

**Clinical Findings** The results produced by the adult worms are variable and include —Lymphangitis Painful red lines form under the skin and the neighbouring lymphatic glands are enlarged There is constitutional disturbance and fever and probably a secondary streptococcal infection of the lymphatics Elephantoid fever Periodical attacks of fever with an initial rigor and terminal sweating occur somewhat resembling malaria The deep lymphatic glands are probably inflamed Other local results of lymphangitis include inflammation of the spermatic cord testicle and synovial membranes Lymphatic varices These may affect the glands especially in the groin (varicose groin glands) They diminish in size on pressure Lymph scrotum is characterised by enlargement of the scrotum with lymphatic varices Ruptured lymphatic varices Chyluria results from rupture in the urinary tract The urine is milky and may contain blood Chylous effusions may form in the peritoneum or in the tunica vaginalis The blood There is an eosinophilia The embryos can usually be seen in a wet film taken at night Elephantiasis In this condition there is a marked solid œdema, due to lymphatic obstruction Microfilariæ are absent from the blood The legs are usually affected, there being an enormous swelling with thickening of the skin and subcutaneous tissues The scrotum may be involved forming a tumour weighing over 50 lbs The vulva and arms are less commonly affected

**Course and Complications** The disease pursues a chronic course Cutaneous carcinoma may occur as a complication of elephantiasis of the leg

**Treatment** *Prophylactic* Mosquito breeding places should be sterilised and electric fans and mosquito nets used as for malaria

*Curative* No specific drug is known but some favourable results have been obtained with Hetrazan It is given by mouth in doses of mg 0 2 to 2 per kg body weight t i d for about 4 to 5 weeks

Acute lymphangitis The limb should be rested and elevated and a lotion applied such as Liq plumb subacetat dil 4 parts alcohol 90% 4 parts aquam ad 100 parts Penicillin or sulphadiazine should be given for secondary streptococcal infections

Chyluria Fat should be omitted from the diet

Elephantiasis An elastic stocking should be worn and some promising results have been obtained by the Kondoléon operation (see p 291) Operation may be required for elephantiasis of the scrotum

*On Examination* The adult worm can usually be seen or felt beneath the skin

**Treatment** *Prophylactic* Small fishes can be placed in water supplies to eat the infected cyclops Water used for drinking or for washing food utensils should be sterilised by boiling

*Curative* The worm may be extracted by injecting with procaine anæsthesia an emulsion along the course of the worm This results in relaxation of the worm so that it can be removed by traction The emulsion consists of phenothiazine 2 G , lanolin 0 35 G , olive oil 35 ml and sterile water 5 ml

# CHAPTER XV

# DISEASES DUE TO PHYSICAL AGENTS

## Caisson Disease

### (*Compressed Air Illness  Diver s Palsy*)

**Definition** A disease due to sudden reduction of atmospheric pressure

**Etiology** This is a disease of workers in caissons who are exposed to compressed air when employed as divers or as builders of bridges tunnels or skyscrapers There is usually no risk unless the pressure is increased by more than that of one atmosphere (+ 15 lb per square inch) The danger is intensified by the length of exposure to the increased pressure Thus divers are less often affected because although when they go to a great depth the pressure in their helmets is high they work at such a pressure for only short periods The symptoms appear on decompression on return to the surface It also affects people flying at 30 000 feet or over

**Pathology** The increased pressure in the caisson causes excess of oxygen and nitrogen to be taken up by the blood The oxygen combines with the tissues and does no harm The nitrogen is also absorbed by the tissues especially by fat and by the central nervous system and after a time the blood is completely saturated with nitrogen If the pressure is now rapidly reduced as the worker comes to the surface the nitrogen is liberated from the tissues in the form of bubbles these bubbles may appear in the fatty tissues spinal cord brain intestines and also in the blood Thus the capillaries may be obstructed by gas emboli or the heart filled with gas Permanent damage may be done to tissues by thus cutting off their blood supply Fat people absorb more nitrogen and so are more prone to the disease Post mortem Numerous gas bubbles may be seen in the brain the cord especially in the lower thoracic portion in the subcutaneous tissues the heart and in certain viscera such as the liver

**Clinical Findings** The patient usually gives a history of a rapid return to the surface from a depth and notices symptoms about ½ to 3 hours later In mild cases there is headache with pains in the joints especially the knees and ankles and in muscles (the bends ) The legs are usually affected and the joints are kept flexed In more severe instances there is giddiness (the staggers ) abdominal pain nausea vomiting shortness of breath (the chokes ) intense itching of the skin (the itch ) and paralysis usually paraplegia or collapse and death may rapidly ensue

**Differential Diagnosis** The history of the case and clinical picture render the diagnosis obvious In some cases the patient is first seen in coma and in others the condition may suggest an acute abdominal lesion

**Course and Complications** Unless adequate treatment is given at

once, permanent nervous damage or death may ensue Secondary hæmorrhage may occur in the affected tissues Chronic arthritis and aseptic bone necrosis may develop as complications

**Prognosis** This depends entirely upon the severity of the condition and the means available for early treatment

**Treatment** *Prophylactic* Fat men, and those suffering from diseases of the heart lungs or kidneys or men addicted to alcohol must not be employed as caisson workers Inflammation of the Eustachian tubes is also a contra indication as there is a risk of rupture of the tympanic membrane owing to inability to equalise the pressure in the middle ear by swallowing movements during the time the external pressure is being raised Men should not work longer than a 1 hour shift at a pressure of + 50 lb longer shifts are permissible for lower pressures When working at increased pressures decompression must be gradual The men pass through a series of air locked chambers where the pressures are gradually lowered a due stay being enforced in each chamber Exercise and the inhalation of oxygen are also important during the decompression

*Curative The patient should be placed in a chamber ( medical air* lock ) and the pressure raised to that at which he was working After half an hour he is very gradually decompressed If no chamber is available the patient may be lowered to the pressure at which he was originally working and gradually brought to the surface If this is not feasible oxygen and morphine should be administered

## Mountain Sickness

### (*Anoxæmia*)

**Definition** Illness resulting from exposure to a low barometric pressure

**Etiology** Mountain sickness results from ascents to great heights, usually over 12 000 feet as in climbing or flying

**Pathology** There is a deficient supply of oxygen to the blood Compensatory changes may occur such as increase in the number of red cells in the blood (erythrocytosis) there are no nucleated red cells, but the reticulocytes are more numerous There is alkalæmia with a fall in the alkali reserve

**Clinical Findings** In rapid flying ascents to over 25 000 feet death may occur after a preliminary stage of dulling of all the mental faculties followed by muscular paralyses In more gradual ascents the patient complains of headache muscular weakness giddiness, palpitations dyspnœa nausea vomiting and fainting attacks He appears cyanosed

**Treatment** A portable oxygen apparatus should be used at high altitudes

## Sea Sickness

### (*including Train Car and Air Sickness*)

**Definition** Sickness occurring on the sea in a train a car or an aeroplane

**Etiology** There are various theories as to the causation of these varieties of sickness such as Labyrinthine, ocular or splanchnic disturbances a neurosis, resulting from auto suggestion acidosis and hypoglycæmia Train car and air sickness are probably closely allied conditions *Predisposing causes* 1 Age Infants and the aged are immune all other ages are susceptible 2 Those subject to migraine or suffering from diabetes mellitus or nephritis and pregnant women are especially liable

**Pathology** It has been shown that before vomiting occurs there is an increased output of ammonia in the urine with acetone bodies There is also a preliminary hyperglycæmia followed by hypoglycæmia Acidosis is more severe when the vomiting stage is reached

**Clinical Findings** The unfortunate victim feels squeamish with a tendency to yawn and salivate He may have headache and dis turbance of vision such as diplopia The vomiting is usually ushered in by more profuse salivation in severe cases there is marked collapse the sufferer loses interest in life and wishes for a speedy end to his torments The face is pale or greenish the skin is cold the pulse rapid and blood pressure low The output of urine is diminished and diarrhœa or constipation may occur

**Differential Diagnosis** It is important not to overlook any abdominal condition such as acute appendicitis intestinal obstruction or a perforated gastric ulcer Attention must be paid to the presence of abdominal rigidity in organic abdominal lesions and alterations in temperature and pulse rate should be recorded on an hourly chart The result of enemas is of diagnostic value in intestinal obstruction

**Course and Complications.** Some individuals are never able to overcome sea sickness in others accommodation is rapidly established Concentration of the urine may lead to bladder irritability resembling that due to cystitis

**Prognosis** Sea sickness is never fatal pregnant women do not miscarry

**Treatment** A meal rich in carbohydrate should be taken before the voyage and the blood sugar kept up subsequently by eating lump sugar at the earliest appearance of symptoms Fats should be avoided As a prophylactic a Marzine (cyclizine hydrochlorid ) tab mg 50 may be taken half an hour before departure and repeated if necessary one to three times daily

## Heat stroke

(*Sunstroke Heat Hyperpyrexia*)

**Definition** Illness resulting from exposure to the sun

**Etiology** Heat stroke usually results from exposure to the sun It generally occurs in the tropics when the shade temperature reaches 110 F White races are very susceptible *Predisposing causes* Debility from other diseases alcoholism and constipation There is often an associated infection such as a boil malaria or gastro enteritis

**Pathology** The heat regulating mechanism is disturbed Post

*mortem, the right heart is dilated and there is œdema of the central* nervous system

**Clinical Findings** The patient gives a history of exposure to *sunlight* He is suddenly taken ill with headache, and may vomit and have *diarrhœa* The patient collapses and may rapidly become unconscious

*On Examination* The face is flushed the skin is hot and dry, the *pupils* are dilated the pulse and respirations are rapid and the temperature is raised *to 109° F or higher* Convulsions may occur The knee jerks are absent The cerebro spinal fluid pressure is usually raised, and there may be an increase first of the polymorphonuclear cells and later of the lymphocytes The breathing may become irregular of the Cheyne Stokes type, and the pupils are constricted before death

***Differential Diagnosis*** A blood film should be taken to exclude malaria

**Course and Complications** Death may rapidly ensue Complications include dilatation of the right heart and œdema of the lungs If the patient recovers from coma there may be mental confusion aphasia incontinence and signs of a pyramidal or cerebellar lesion Complete recovery may ensue or the patient may show signs of dementia

**Treatment** *Prophylactic* The bowels should be kept open daily plenty of fluid consumed, but alcohol should not be taken to excess The head, neck and spinal cord should be adequately protected from the sun

*Curative* The temperature is lowered by applying ice to the head *neck and spine total immersion in ice cold water with massage of the* extremities spraying the body with ice cold water, and if necessary by giving a rectal injection of ice cold water At the same time a current of air should play on the body produced either by electric fans or by flapping towels When the rectal temperature falls to 102° F these measures must be discontinued Venesection should be performed if the patient is cyanosed Lumbar puncture may be required to relieve increased cerebro spinal fluid pressure An intravenous injection of quinin dihydrochlor gr 10 in 20 ml of distilled water should be given slowly if there is any doubt as to the condition being due to malaria

## Heat Exhaustion and Heat Cramp

**Definition** Illness resulting from exposure to heat

**Etiology** Heat exhaustion is prone to develop in hot moist atmospheres when evaporation from the skin is low Heat cramp results from exposure to dry heat, the excessive sweating leading to diminution of the blood chlorides Stokers miners and iron and steel workers are subject to heat exhaustion without any direct exposure to sunlight

**Clinical Findings** The patient has usually been working in a very hot atmosphere *such as that which a stoker must endure* He becomes weak, giddy, and may sweat and collapse In some instances there is vomiting and diarrhœa or painful cramps may be felt in the legs

*On Examination* In heat exhaustion the patient is usually pale owing to vaso constriction the skin is moist and the oral temperature may be subnormal whereas the rectal temperature is raised to about 102° F The skin and subcutaneous tissues show evidence of dehydration In more severe cases there is marked shock the pulse is frequent and feeble and unconsciousness may ensue The blood pressure is low Uræmic symptoms may result from renal anoxia The urine output is low the urine may contain casts and the sodium chloride content is much reduced Heat cramp may also occur in temperate climates

**Treatment.** The patient should be moved to a cool place and treatment directed to correcting the dehydration sodium chloride loss and shock A careful record should be kept of the fluid intake and output and the urinary chlorides estimated every 8 hours In severe cases an intravenous injection of normal saline should be given 1 000 ml or more according to the dehydration and chloride content of the urine Half normal saline may also be given by mouth or by nasal intra gastric drip Heat cramp may be prevented or relieved by drinking 0 1% sod chlorid solution in orangeade or by taking by mouth tablets of sod chlorid, gr 15 up to 15 or 20 in the day

## Frost Bite

**Definition** The harmful effects produced by cold on peripheral parts of the body Under the term the cryopathies are included frost bite immersion foot and hand trench and shelter foot

**Etiology** Frost bite results from exposure to severe cold especially if the individual is in a high wind or is at a great altitude Trench foot is a variety of frost bite The causative factors here are cold not necessarily of a severe degree stagnation of circulation due to standing in wet and muddy trenches ill fitting boots and tight clothing on the legs

**Pathology** Severe cold produces peripheral cutaneous vaso constriction followed by liberation of H substance vasodilatation damage to vessels and transudation of fluid into the surrounding tissues This results in œdema and bulla formation In more severe cases of frost bite thrombosis and gangrene ensue The effect of high altitude and oxygen lack is an increase of pulse rate up to 180 to 250 per minute with a reduction in the output (minute volume) of blood from the heart

**Clinical Findings** Frost bite may occur very rapidly *e g* if a metallic object is touched by the tip of a bare finger in severe cold or at a high altitude In other cases where the exposure is more prolonged as may happen when tramps sleep out in the winter there is first a feeling of burning in the extremities followed by a lack of all sensation The onset of trench foot is much more insidious taking often days or weeks

*On Examination* Various stages of frost bite may be seen such as a white and waxy appearance of the skin in mild cases In more severe ones the skin is red or almost black with bulla formation and gangrene Trench feet are usually bluish red Varying degrees of hypoæsthesia to

light touch, pin prick and temperature sensation are present in frost bite and trench foot

**Course and Complications** *Pain is experienced in the extremities* as recovery takes place The nails may be shed or portions of the extremities lost by gangrene

**Treatment** *Prophylactic* Protection by means of adequate clothing dry gloves and ear flaps, is important Properly fitting supple well oiled boots must be supplied to soldiers in trenches Puttees and darned socks should not be worn and socks should be changed when wet as soon as feasible *The body temperature should be kept as warm as* possible

*Curative* If a foot or hand becomes numb or the nose goes white the affected part should be warmed by taking off the boot or glove and placing the limb between the thighs or under the arm of a comrade The nose should be warmed between the hands In more advanced cases local application of heat in any form must be avoided and friction which includes rubbing with snow, must on no account be applied to the affected parts These should be cleaned gently, using gauze soaked in 1/1 000 proflavine then carefully dried and covered with sterile gauze and several layers of wool Bullæ which have burst or gangrenous areas should be dusted with sulphathiazole powder General treatment consists in combating shock by the administration of hot drinks If the body temperature is subnormal the bed should be warmed with hot bottles which must not be placed near the affected parts Prophylactic injections of antitetanic serum should be given and anticoagulants (see p 187) to minimise the risk of thrombosis Antibiotics should be used to prevent infection In cases developing at high altitudes the administration of *oxygen is of great value* *Amputation may be required* for gangrene but this should be delayed as long as possible as partial recovery is likely to ensue For the later persistent symptoms pain swelling loss of movement the Raynaud phenomenon and hyper hidrosis due to vascular occlusion and resembling those met with in thrombo angiitis obliterans preganglionic sympathectomy may afford relief

## Electric Shock and Burns

**Definition** The harmful effects produced by electric currents

**Etiology** Electric shock is usually accidental as by contact with a live wire on an electric railway or tramway system *by touching* an electric heater when in a bathroom, or by contact with a house lighting system Death in the bathroom is due to a fault in the electric system whereby some of the current is entering the switch The victim is usually in the bath which acts as a condenser, and on switching off the heater his body is subjected to a high current The result is not usually fatal with voltages under 300 for the direct current or under 100 with an alternating current Very high voltages of alternating current, on the other hand may do little or no harm Lightning may cause electric shock and burns may occur from X rays or diathermy used in medical treatment In the lightning flash there is a rush of

protons and electrons through the air the current is direct and the voltage very high 1 000 volts or more with perhaps 20 000 amperes

**Pathology** The body is often charred locally, the blood is fluid and small hæmorrhages may be found in the brain The heart may be arrested by ventricular fibrillation this causes death in 90% of cases In death from lightning an effusion is found under the scalp without any superficial abrasion Burns of varying degree are seen on the body

**Clinical Findings** The patient is often killed outright when struck by lightning the clothes may be stripped off the body In other cases the victim is rendered unconscious but slowly recovers experiencing great pain as the circulation is restored In slighter degrees of electric shock the patient who has gripped a live wire is unable to relax his hold is terrified and feels severe pain Electrical burns of all kinds are very slow in healing Albuminuria may be noted subsequently due to the presence in the blood of abnormal protein substances

**Treatment** The current should be switched off and the patient removed If the current cannot be cut off the hands of the rescuer should be protected by rubber gloves or some dry thick material He should also stand on a dry substance such as bricks or cloth Artificial respiration should be applied for several hours if the patient is unconscious The Eve rocking method appears to ventilate the lungs better than Schafer s method The patient is placed face downwards on a stretcher well wrapped up the ankles and wrists bandaged to the handles the stretcher being rested on a trestle 3 feet high and the rocking should be a full head down tilt of 45 When fluid has ceased to run from the lungs and stomach rocking is begun with 10 double rocks every minute 4 seconds with the head down and 2 seconds with the feet down In addition Coramine 5 to 10 ml should be injected intravenously

# CHAPTER XVI

# THE POISONS

**Introductory** Acute poisoning is usually due to substances taken by inhalation or by mouth rarely by injection *Carbon monoxide is the* most common cause of fatal poisoning in England and in America and the treatment by artificial respiration and the inhalation of carbon dioxide and oxygen is considered on p 782 To rid the body of swallowed poisons which have not yet been absorbed gastric lavage is of the greatest value Emetics should only be given if the requisites for gastric lavage are not available Purges are seldom required Gastric lavage is contraindicated if strong corrosives, such as strong nitric sulphuric or hydrochloric acid caustic potash or soda, or strong ammonia have been swallowed In such cases the corrosive should be neutralised Strong acids can be neutralised with four tablespoonfuls of light or heavy magnesia to a pint of water half a pint being used for a child To neutralise strong alkalis six tablespoonfuls of vinegar or the juice of six lemons are added to a pint of water Apart from poisoning by such strong corrosives it is always advisable to wash out the stomach even if the patient has vomited or if several hours have passed since the poison was taken Gastric lavage can be safely used in the case of poisoning by oxalic or carbolic acid or by Lysol

Very thorough lavage should be given, using 2 gallons of warm water Great care should be taken to prevent the fluid from regurgitating into the lungs and for this purpose the method recommended by Marriott should be followed The patient is placed prone with his head hanging over the end of the couch or bed the forehead being *supported by an assistant A Jaques firm rubber stomach* tube (œsophageal tube) size 23–30 English catheter gauge 60 inches long *is used for an adult A safety pin is passed through* the wall not the lumen of the tube, 20 inches from the end which will lie in the *stomach* Any *false* teeth having been removed and the mouth being kept open by a gag the lubricated tube is passed into the mouth until it reaches the pharyngeal wall and is then pushed quickly down into the stomach The safety pin in the tube should lie about ½ inch outside the incisor teeth The tube is passed with the patient in the prone position the operator sitting or kneeling on the floor to do so A funnel is attached to the upper end of the tube and a pint of warm water run into the stomach The funnel is then inverted over a pail on the *floor* and the stomach contents are syphoned out In this way 2 gallons of water are used for the lavage Care must be taken that *the tube does* not alter its position while the lavage is being carried out If emetics are required a subcutaneous injection of apomorphin *hydrochlor gr 1/10* will produce vomiting in 5 minutes Other emetics which may be used include pulv ipecac *gr 30, mustard or salt ¼ oz* in a tumbler of warm

water Dehydration and loss of chlorides are liable to occur if there has been severe vomiting and diarrhœa If the patient can swallow he should be given half normal saline (NaCl gr 40 to a pint) sweetened with dextrose 1 oz. and the juice of two oranges If he cannot swallow normal saline should be given by drip transfusion either rectally or intravenously Sufficient fluid should be administered to relieve thirst, improve the blood pressure and increase the urinary output but care must be taken not to give sufficient to embarrass the heart or cause œdema of the lungs

## Mercury Poisoning

**Etiology** Mercurial poisoning may occur 1 As an occupational disease, in miners of mercury, thermometer, barometer or looking glass makers and felt hat makers who use mercury nitrate 2 If mercury (usually the perchloride) is taken for suicidal or homicidal reasons 3 In accidental contamination of foods or by accidentally drinking perchloride of mercury 4 In the therapeutic use of mercury, due to gradual overdosage or personal idiosyncrasy

**Pathology** In acute poisoning there is found post mortem intense inflammation of the mucous membrane of the stomach and intestines The stomach mucous membrane may be greyish white and a greyish membrane may form in the large intestine The kidneys show the changes of acute tubular necrosis

**Clinical Findings** *Acute Poisoning* This usually results from taking perchloride of mercury solution by mouth either accidentally or suicidally or by using it as a vaginal douche Within a few minutes of ingestion there are severe epigastric pains and vomiting The vomit may contain blood Diarrhœa rapidly follows and the stools also may be bloodstained Prostration and collapse follow according to the severity of the intoxication The blood This may show an increase of urea and non protein nitrogen The urine This is highly acid and may contain albumin blood and casts Suppression of urine may ensue

*Chronic Poisoning* Therapeutic overdosage with mercury is characterised by salivation a metallic taste stomatitis gingivitis offensive breath and diarrhœa In industrial poisoning there may be also headache, lassitude anæmia loosening of teeth albuminuria a rise of blood pressure and marked tremors of an intentional variety Mercurial dermatitis with erythema and desquamation may appear Mercurial erythism is sometimes noted and is characterised by excitability shyness with strangers insomnia depression and giddiness

**Differential Diagnosis** The diagnosis is usually apparent from the history and typical clinical findings Mercury may be present in the fæces

**Course and Complications** The course depends upon the amount of mercury ingested Complications include renal tubular necrosis anæmia and colitis

**Prognosis** This is very grave in acute cases but has been much improved by modern treatment. Death may occur in a few hours or

days In chronic cases recovery usually results and depends upon the rapidity of the recognition of intoxication and withdrawal of the poison Tremors may persist for several years

**Treatment** *Acute Poisoning* The stomach should be washed out with 1 gallon of a warm saturated solution of sodium bicarbonate and 8 oz of a saturated solution of magnesium sulphate left in the stomach A soap and water enema should then be given Alternatively the stomach may be washed out with 250 ml of 5% sod formaldehyde sulphoxylate B A L is then injected in the doses given on p 546 For relief of the abdominal pain and diarrhœa tnc opii m 30 should be given 2 or 3 times daily

*Chronic Poisoning* *Prophylactic* Workers in a dangerous occupation should be subjected to periodical medical examinations When mercury is being administered a watch should be kept constantly for early signs of mercuralism

*Curative* The mercury administration must cease or the worker be removed from exposure to the poison Elimination is aided by administering mag sulph gr 60 to 120 daily For mercurial dermatitis a lotion of 1% sodium hyposulphite should be applied (gr 4 to fl oz 1)

## Lead Poisoning

### (*Plumbism* *Saturnism*)

**Etiology** Lead may enter the body through the alimentary tract it may be inhaled as dust, or absorbed through abrasions in the skin or mucous membranes Lead poisoning is chiefly of importance as an occupational disease The following are the chief causes 1 *Occupations* Workers in red and white lead painters potters brass founders miners rubber mixers, accumulator manufacturers and printers, and those engaged in the manufacture of tetra ethyl lead and in blending it with petrol 2 *Food* Soft water may be contaminated by lead pipes beer may be similarly affected, cider may be tainted by lead glaze in jars and wine by subacetate of lead added as a sweetening agent Tinned foods may cause poisoning, especially oily fish in soldered tins and illness has resulted from eating cakes coloured yellow with lead chromate 3 *Cosmetics* Hair dyes toilet powders or face creams may contain lead 4 *Drugs* Ointments containing lead may cause poisoning when applied to broken surfaces or lead lotions used as an eye or vaginal douche Lead pills made from diachylon plaster were formerly used to obtain abortion but the sale has been checked by the Poisons Act Lead tetra ethyl can be absorbed through the skin and the vapour given off is absorbed freely through the lungs and, owing to its fat soluble properties, it poisons especially the central nervous system *Predisposing causes* Women and children are more susceptible than men, and negroes than white men Debility from any acute illness and chronic alcoholism are also predisposing factors

**Pathology** Lead absorbed from the alimentary canal is carried to the liver and may then pass into the systemic circulation or be excreted in the bile It is conveyed by the blood stream to all the tissues

of the body but is stored especially in the bones It circulates in the blood plasma as a colloidal phosphate and accumulates in the solid parts of the bones probably as an insoluble triple phosphate $Pb_3(PO_4)_2$ It may be liberated from the bones and flood the blood It is excreted chiefly in the fæces to a slight degree in the urine and less still by the skin Inhaled lead dust is carried direct to the systemic circulation A slight increase of the acidity or alkalinity of the blood appears to result in liberation of lead from the bones as does also any agent which causes solution of calcium salts from the bones Post mortem Gastro-enteritis is found in acute lead poisoning in chronic cases nervous lesions such as degeneration of anterior horn cells peripheral neuritis and muscular atrophy may be found The amount of lead present in the skeleton varies between 0 2 and 0 8 G Such lesions as arteriosclerosis and chronic nephritis are not indubitably due to lead

Clinical Findings *Acute Lead Poisoning* This may be a primary disease caused by a large dose of lead taken with suicidal intent or accidentally If a large dose of lead is swallowed the patient suffers from burning in the mouth thirst dysphagia intestinal colic vomiting cramps in the legs and convulsions The bowels are constipated Acute symptoms or ' toxic episodes may also occur during the course of chronic lead poisoning and these are described later

*Subacute and Chronic Lead Poisoning* Twenty five cases of tetra ethyl lead poisoning 2 of which were fatal occurred in this country in 1945 during tank cleaning operations The tanks had been used for storing aviation petrol the sludge at the bottom of the tanks containing organic lead compounds Further the majority of the tanks were underground The early symptoms were difficulty in getting to sleep troubled dreams a nasty sweet taste halitosis anorexia diarrhœa with or without abdominal pain trembling giddiness and loss of weight There was no basophilic stippling of the red cells In other cases the worker complains of lassitude and dyspnœa on exertion He becomes constipated loses his power of concentration has vague pains in the arms and shoulders and loses weight Cramps may occur in the legs Periodically acute symptoms known as toxic episodes may occur Thus the subject of chronic plumbism may have severe intestinal colic This usually follows a period of marked constipation the pain is generally hypogastric and may last for several days Cramp may also be acute in the legs bladder or uterus with menorrhagia or abortion if the patient is pregnant Acute nervous symptoms such as mania or convul sions coma or delirium may develop due to a meningo encephalopathy Other nervous le ions may ensue more insidiously such as optic neuritis and optic atrophy with resulting blindness or the worker may experience difficulty in using the muscles especially concerned with the performance of his duties Temporary blindness may result from retinal vascular spasm Uræmic symptoms may be due to renal anoxia secondary to vascular spasm

*On Examination* The patient is pale and the skin has a greyish tinge The teeth are often carious and pyorrhœa is present A blue line may be seen on the gums adjacent to carious teeth or on the mucous

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**Etiology** Lead may enter the body through the alimentary tract it may be inhaled as dust or absorbed through abrasions in the skin or mucous membranes Lead poisoning is chiefly of importance as an occupational disease The following are the chief causes 1 *Occupations* Workers in red and white lead painters, potters brass founders miners, rubber mixers, accumulator manufacturers and printers, and those engaged in the manufacture of tetra ethyl lead and in blending it with petrol 2 *Food* Soft water may be contaminated by lead pipes beer may be similarly affected cider may be tainted by lead glaze in jars and wine by subacetate of lead added as a sweetening agent Tinned foods may cause poisoning, especially oily fish in soldered tins and illness has resulted from eating cakes coloured yellow with lead chromate 3 *Cosmetics* Hair dyes toilet powders or face creams may contain lead 4 *Drugs* Ointments containing lead may cause poisoning when applied to broken surfaces or lead lotions used as an eye or vaginal douche Lead pills made from diachylon plaster were formerly used to obtain abortion, but the sale has been checked by the Poisons Act Lead tetra ethyl can be absorbed through the skin and the vapour given off is absorbed freely through the lungs and owing to its fat soluble properties it poisons especially the central nervous system *Predisposing causes* Women and children are more susceptible than men, and negroes than white men Debility from any acute illness and chronic alcoholism are also predisposing factors

**Pathology** Lead absorbed from the alimentary canal is carried to the liver and may then pass into the systemic circulation or be excreted in the bile It is conveyed by the blood stream to all the tissues

of the body, but is stored especially in the bones It circulates in the blood plasma as a colloidal phosphate and accumulates in the solid parts of the bones probably as an insoluble triple phosphate $Pb_3(PO_4)_2$ It may be liberated from the bones and flood the blood It is excreted chiefly in the fæces to a slight degree in the urine and less still by the skin Inhaled lead dust is carried direct to the systemic circulation A slight increase of the acidity or alkalinity of the blood appears to result in liberation of lead from the bones as does also any agent which causes solution of calcium salts from the bones Post mortem Gastro-enteritis is found in acute lead poisoning in chronic cases nervous lesions such as degeneration of anterior horn cells peripheral neuritis and muscular atrophy may be found The amount of lead present in the skeleton varies between 0 2 and 0 8 G Such lesions as arteriosclerosis and chronic nephritis are not indubitably due to lead

Clinical Findings *Acute Lead Poisoning* This may be a primary disease caused by a large dose of lead taken with suicidal intent or accidentally If a large dose of lead is swallowed the patient suffers from burning in the mouth thirst dysphagia intestinal colic vomiting cramps in the legs and convulsions The bowels are constipated Acute symptoms or toxic episodes may also occur during the course of chronic lead poisoning and these are described later

*Subacute and Chronic Lead Poisoning* Twenty five cases of tetra ethyl lead poisoning 2 of which were fatal occurred in this country in 1945 during tank cleaning operations The tanks had been used for storing aviation petrol the sludge at the bottom of the tanks containing organic lead compounds Further the majority of the tanks were underground The early symptoms were difficulty in getting to sleep troubled dreams a nasty sweet taste halitosis anorexia diarrhœa with or without abdominal pain trembling giddiness and loss of weight There was no basophilic stippling of the red cells In other cases the worker complains of lassitude and dyspnœa on exertion He becomes constipated loses his power of concentration has vague pains in the arms and shoulders and loses weight Cramps may occur in the legs Periodically acute symptoms known as ' toxic episodes may occur Thus the subject of chronic plumbism may have severe intestinal colic This usually follows a period of marked constipation the pain is generally hypogastric and may last for several days Cramp may also be acute in the legs bladder or uterus with menorrhagia or abortion if the patient is pregnant Acute nervous symptoms such as mania or convul sions coma or delirium may develop due to a meningo-encephalopathy Other nervous le ions may ensue more insidiously such as optic neuritis and optic atrophy with resulting blindness or the worker may experience difficulty in using the muscles especially concerned with the performance of his duties Temporary blindness may result from retinal vascular spasm Uræmic symptoms may be due to renal anoxia secondary to vascular spasm

*On Examination* The patient is pale and the skin has a greyish tinge The teeth are often carious and pyorrhœa is present A blue line may be seen on the gums adjacent to carious teeth or on the mucous

membrane of the cheek or lips opposite the bad teeth It was described by Burton in 1840 This line consists of a series of blue black dots, best seen with a hand lens situated just below the margin of the gums and in the subepithelial tissue It is due to lead sulphide the $H_2S$ being formed by putrefaction in the mouth Tremors of the tongue and hands may be noted Peripheral nerve lesions are generally bilateral They include Antebrachial paralysis with wrist drop, due to paralysis of the posterior interosseous branch of the musculo spiral nerve the supinator longus muscle is therefore spared Aran Duchenne paralysis, with wasting of the small muscles of the hand and thenar and hypothenar eminences a claw hand ' resulting Brachial paralysis, the biceps supinator longus brachialis anticus and deltoid muscles are affected Peroneal paralysis with foot drop and paralysis of the peroneal muscles extensor longus digitorum and extensor proprius hallucis Recurrent laryngeal nerve paralysis with adductor cord paralysis, may occur In some instances there is a diffuse muscular paralysis and in others a cerebellar ataxia appears Sensory changes are generally absent The reaction of degeneration is obtained in the affected nerves

*Examination of a Patient during an Attack of Colic* The pain is obviously very severe the patient is pale sweating and holds his hand to his abdomen as pressure affords some relief the abdominal wall is not really rigid and is hollow rather than distended The temperature is subnormal and the pulse slow The blood There is an anæmia The red cells show stippling " (punctate basophilia) and at least 100 red cells per million red cells must be so stippled to be diagnostic These stippled cells are reticulocytes The white cells are usually normal The fæces These usually contain lead as does the urine if lead has reached the blood stream The cerebro spinal fluid In the convulsive stage it is under pressure and contains an excess of lymphocytes

**Differential Diagnosis** It should be remembered that the symptoms of plumbism may develop after an individual has ceased to be exposed to lead Various factors may cause lead to be liberated from the bones into the blood The diagnostic features are the blue line the punctate basophilia and the presence of lead in the urine As traces of lead may be found in the urine in health, due to lead eaten with food at least 0 1 to 0 8 mg lead per litre of urine must be present to be diagnostic

**Course and Complications** The course is characterised by the "toxic episodes ' described above Complications include such con ditions as arteriosclerosis, chronic nephritis and gout

**Prognosis** Recovery is apt to be slow Colic usually rapidly responds to appropriate treatment paralyses tend to persist unless exposure to lead is stopped at the earliest symptom recovery occurring first in the muscles last affected Death is especially liable to occur in maniacal cases

**Treatment** *Prophylactic* Measures should be taken to prevent workmen inhaling lead dust these include the use of wet processes and the employment of adequate fans and respirators Workers in lead should wash their hands before eating and change their clothes on leaving work Periodical medical examinations at least monthly should

be held to detect early signs of plumbism, and any suspicious cases suspended from work and kept under observation The urine should be examined for its lead content. If the lead rises to between 0 15 and 0 25 mg per litre it is probable that poisoning is taking place A high calcium diet such as one containing one to two pints of milk daily should be taken

*Curative* In acute poisoning accidental or suicidal the stomach should be emptied by an emetic such as mustard, ½ oz in ½ pint of warm water or preferably washed out with 2 gallons of warm water containing 2 oz. of mag sulph and a saline aperient is then given, such as Mag sulph. sod sulph aa gr 240 acid sulph dil m 30 aquam ad fl oz. 10 Fl oz. 10 to be taken every 4 hours until the bowels are thoroughly evacuated For abdominal colic hot flannels should be applied locally and an intravenous injection given slowly of 10 ml of 20% calcium gluconate In the acute exacerbations of chronic lead poisoning efforts are directed to retain lead in the bones by giving a diet rich in calcium such as milk 4 pints in 24 hours together with calcium lactate gr 30 t d s by mouth.

Lead can be removed from the body and excreted in the urine by the intravenous injection of calcium disodium versenate 0 5 G per kg body weight being injected every 12 hours The calcium disodium versenate is introduced into 300 ml 5% dextrose solution for each intravenous injection Four or five courses each lasting 3 or 4 days are given In children lead is liable to remain in the bones which may give rise to recurrent episodes of lead poisoning The resulting anæmia should be treated with iron or by blood transfusion

## Arsenic Poisoning

Etiology Arsenical poisoning may be due to 1 Occupations such as extracting white arsenic from arsenical pyrites or preparing sheep-dip Arsine may be inhaled in chemical works or in submarines where it is liberated from battery plates 2 Administration of arsenic with homicidal or suicidal intent Weed killers fly papers or rat pastes may be used 3 Food stuffs accidentally contaminated such as glucose in the preparation of beer the sugar covering sweets and the skin of apples which have been sprayed with an arsenic solution as a parasiticide In 1943 there was an outbreak in St Andrews of 150 cases of acute arsenical poisoning with 2 deaths following on the consumption of sausages Arsenious oxide was recovered from the sausage meat but the source of the arsenic was not traced 4 Therapeutic administration of arsenic either due to an overdose being given or to idiosyncrasy of the patient It may occur with such preparations as Fowler s solution sodium cacodylate Atoxyl tryparsamide or neoarsphenamine.

Pathology In acute cases there is inflammation of the mucous membrane of the stomach and upper part of the small intestine and cloudy swelling may be found in the liver, heart and kidneys In cases of chronic poisoning fatty degeneration is present in most of the

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**Pathology** In acute cases there is inflammation of the mucous *membrane of the stomach and upper part of the small* intestine and cloudy swelling may be found in the liver, heart and kidneys In *cases of chronic poisoning fatty degeneration is present in most of the*

organs of the body Arsenic can be detected in the body for many months after death

**Clinical Findings** *Acute Poisoning* Directly after taking a poisonous dose the patient experiences dryness in the throat and this is soon followed by a burning sensation in the epigastrium He feels sick and vomits Intestinal colic and severe diarrhœa usually follow and there may be cramps in the legs Small amounts of blood may be present in the vomit or the stools

*On Examination* In a severe case the patient is cold and collapsed the pulse rapid and of small volume

*Chronic Poisoning* The symptoms here are insidious In the early stages there is irritation of the nasal and conjunctival mucous membranes and later of the pharynx and larynx If arsenic is being given as medicine these symptoms should be a sufficient warning of an overdosage Further administration of arsenic leads to abdominal discomfort, loss of appetite, nausea, vomiting tetany, intestinal colic and diarrhœa The tongue has a silvery white fur There is a generalised pigmentation of the skin especially in the flexures where ulceration may occur Keratosis may develop on the soles and palms Arsenical neuritis causes pains in the legs and arms cramps and paresis of the legs Herpes simplex may develop Sudden blindness due to optic atrophy may occur with Atoxyl or tryparsamide Arsenic may be found in the urine fæces and vomit and also in the nails or hair

**Differential Diagnosis** In general practice it is a delicate matter to reveal one s suspicions of arsenic poisoning but it is the duty of the doctor to have the urine, fæces nails or hair examined in any doubtful case Acute arsenical poisoning may be mistaken for cholera or other forms of acute gastro enteritis Chronic arsenical poisoning may be mistaken for gastro enteritis colitis carcinoma of the stomach or Addison's disease

**Course and Complications** These depend on whether or not arsenic is continuously administered In progressive cases death usually occurs from heart failure with ascites and œdema

**Prognosis** If fatal death from acute arsenical poisoning usually occurs in a few hours or days The prognosis in chronic cases depends upon the recognition of the disease and the cessation of the administration of arsenic

**Treatment.** *Acute Poisoning* The stomach should be emptied by giving an emetic, such as mustard (½ oz in ½ pint of warm water) until vomiting occurs or preferably by gastric lavage Freshly prepared ferric hydroxide should be used in gastric lavage as this forms an insoluble arsenite It is made by adding sod bicarb to liq ferri perchlor fl oz 2 until effervescence ceases The precipitate of ferric hydroxide is filtered off and added to 2 gallons of warm water An aperient should be given after an hour consisting of mag sulph gr 240 in half a tumbler of water General treatment consists in keeping the patient warm administering stimulants such as digitalin gr 1/100 hypodermically and if the abdominal pain is very severe a hypodermic injection of morphin sulph gr 1/6 to 1/4 may be given

B A L should be given both for acute and chronic arsenical poisoning in doses recommended on p 516

In chronic poisoning after discontinuation of the arsenic the treatment is symptomatic for the debility and neuritis

## Alcoholic Poisoning

**Etiology** Alcohol in any form may cause symptoms of poisoning It is usually due to ethyl alcohol less frequently to methyl alcohol *Predisposing causes* 1 Occupation Public house keepers barmen and commercial travellers *2 Heredity A neuropathic predisposition* 3 Worry and pain The habit may persist after alcohol has been ordered by a doctor during illness

## Acute Alcoholic Poisoning

(*Physiological Inebriation*)

**Pathology** *Alcohol is rapidly absorbed from the stomach and* intestines and passes to the blood and cerebro spinal fluid Post mortem there may be hyperæmia of the gastric mucous membrane and of the brain

**Clinical Findings** The patient suffering from acute alcoholic poisoning is drunk but the definition of drunkenness from the medico-legal aspect has not been satisfactorily established A person is usually considered to be drunk when as a result of taking alcohol he is not able to perform with his normal skill or facility the ordinary actions of his life The higher centres are first inhibited and then the lower In the early stages there is diminution of cerebral restraint The individual loses any sense of shyness and may become talkative or emotional The onset of muscle fatigue is delayed The finer acts of co-ordination are disturbed and later there is unsteadiness of gait inability to walk and finally coma In some instances there is no excited stage but marked depression from the onset

*On Examination* The breath smells of alcohol the face is usually flushed but may be pale the skin is moist the pupils dilated and the pulse rapid There is difficulty in the pronunciation of such words as British Constitution and inability to walk steadily along a line In alcoholic coma the pupils are dilated the respiration is slow the pulse feeble the body temperature subnormal the limbs flaccid the deep reflexes diminished and the plantar response is flexor The urine The amount of alcohol present in the urine has been suggested as a medico legal test for drunkenness It is said that no man whose urine contains 150 mg alcohol per 100 ml is fit to drive a car The alcohol in the urine bears a direct relationship to the alcohol content of the blood

**Differential Diagnosis** Drunkenness must be distinguished from Excitement due to other causes *Coma due to other causes* such as hypoglycæmia after a dose of insulin The presence of disease or injury in a person who has taken a small amount of alcohol

**Course and Complications** In the majority of cases the patient recovers after a night s rest but alcoholic coma and pulmonary œdema

are always causes of great anxiety  Nephritis or pneumonia may ensue when the patient is exposed to cold of which he is unaware

**Prognosis** This is usually good but death may occur in coma from respiratory or cardiac failure

**Treatment** In the majority of cases no treatment is required beyond an aperient a night's rest in bed and aspirin gr 10 to 15 to relieve the subsequent headache In severe cases the patient should be given an emetic (½ oz mustard in ½ pint of warm water) or preferably gastric lavage should be performed with 2 gallons of warm water containing sodium bicarbonate (1 oz to a pint), and a pint of hot coffee left in the stomach Collapse may be further treated by an electric cradle hot bottles and stimulants such as Coramine 1 5 ml intramuscularly amphetamine sulph mg 10 in 1 ml of isotonic saline intravenously and the intramuscular injection of ephedrine sulph gr ½ If the patient is comatose inhalation of 10% $CO_2$ and 90% oxygen helps to wash out the alcohol and to oxidise it This treatment usually restores the patient to consciousness in half an hour

## Chronic Alcoholic Poisoning

**Pathology** Post mortem there may be fatty infiltration and degeneration of the heart, atheroma of the aorta, fatty infiltration and cirrhosis of the liver dilatation of the œsophageal veins atrophy of the gastric mucous membrane, chronic nephritis of varying types, congestion of the meninges, excess of fluid in the pia arachnoid space ("wet brain") degeneration of cortical motor cells and peripheral neuritis

**Clinical Findings** There are two main types the sober in which intoxication rarely occurs, and the inebriate in which the patient is seldom completely sober The patient may complain that the appetite is poor and there may be morning nausea or vomiting the stools being rather loose Hæmatemesis may occur or bleeding from the rectum due to piles Peripheral neuritis may be the chief complaint The patient is often a woman who suffers from tingling or pains in the calves cramps and weakness of the legs

*On Examination* The patient is often somewhat obese dilated venules are seen on the face the conjunctivæ are watery and injected the tongue is furred and tremulous the throat congested and the breath "heavy" The liver may be enlarged and ascites present

The calves may be tender on deep pressure, and areas of cutaneous anæsthesia may be found over the legs In the early stages of neuritis the knee jerks are increased later they are diminished or absent The leg muscles are weak and may be wasted In some cases in addition to the neuritis there are cerebral symptoms which constitute Korsakow's psychosis The main features are disorientation as regards time and place the patient is unable to remember events which have recently happened such as what he had at his last meal or may say he has been out for a walk that morning when he has been in bed for several weeks Variable oculo motor paralyses and a myotonic pupil may be seen

There are other cases especially in secret drinkers where no local ising effects of the alcohol are present but the patient is unfitted to a varying degree for his work He is irritable lacks concentration and his mentality somewhat resembles that of a child He tries to give up the alcohol but has not sufficient will power

**Course and Complications** The course is usually progressive the patient becoming more and more a slave to alcohol Delirium tremens is liable to occur Pulmonary tuberculosis may follow as a complication

**Prognosis** The outlook is always grave

**Treatment.** The patient should be admitted to an institution devoted to the cure of alcoholism and drug addiction In order to prevent the onset of delirium tremens the alcohol should be ' tapered gradually, and not cut off at once It is given in definite amounts at fixed intervals such as every 2 to 6 hours A reduction of about 2 oz may be made every 24 hours so that at the end of 10 days no alcohol is taken During this stage the patient is best in bed and a mixture is given containing m 5 of tincture of belladonna every 2 hours The dose is gradually increased to a maximum of m 20 every 2 hours Belladonna must not be prescribed if there is a history of glaucoma or if there is considerable bronchitis The dose of belladonna must not be sufficient to *produce dilatation of the pupils Antabuse (tetræthyl thiuram* disulphide) may be given in doses of gr 8 a day and by rendering the patient very sensitive to small doses of alcohol owing to the formation of acetaldehyde it discourages him from taking it It must not be given if the patient is suffering from epilepsy diabetes mellitus cardio vascular or hepatic disease As soon as possible the patient should take a good mixed diet

## Delirium Tremens

Delirium tremens usually occurs in subjects of chronic alcoholism either after an overdose or more commonly when their usual allowance is suddenly cut off as may arise from the patient being put in prison or in a curative institution where the alcohol is suddenly withheld Shock such as an accident and a toxic disease especially lobar pneu monia, when associated with withdrawal of alcohol are important causes The delirium begins with nocturnal visual hallucinations Objects which usually appear blue such as snakes rats or other animals are imagined crawling on the bed or about the room The patient is alarmed and often difficult to restrain Auditory hallucina tions of threatening voices are also heard

**Differential Diagnosis** The onset of delirium tremens may closely resemble that of uræmia owing to the presence of convulsions oliguria albuminuria and urinary casts The blood nitrogen figures are however usually normal but may be raised The temperature may be normal or raised The condition usually passes off in 2 to 3 days the patient sleeping soundly he may however pass into a state of muttering delirium which is followed by death

**Treatment** The patient should be kept in bed in a darkened room and restrained if violent Sedation is effected by giving paraldehyde

are always causes of great anxiety Nephritis or pneumonia may ensue when the patient is exposed to cold of which he is unaware

**Prognosis** This is usually good, but death may occur in coma from respiratory or cardiac failure

**Treatment** In the majority of cases no treatment is required beyond an aperient a night's rest in bed and aspirin gr 10 to 15 to relieve the subsequent headache In severe cases the patient should be given an emetic (½ oz mustard in ½ pint of warm water) or preferably gastric lavage should be performed with 2 gallons of warm water containing sodium bicarbonate (1 oz to a pint) and a pint of hot coffee left in the stomach Collapse may be further treated by an electric cradle hot bottles and stimulants such as Coramine 1 5 ml intramuscularly amphetamine sulph mg 10 in 1 ml of isotonic saline intravenously and the intramuscular injection of ephedrine sulph gr ½ If the patient is comatose inhalation of 10% $CO_2$ and 90% oxygen helps to wash out the alcohol and to oxidise it This treatment usually restores the patient to consciousness in half an hour

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## Carbon Monoxide Poisoning

**Etiology** Carbon monoxide is present to the extent of about 15% in coal gas It is also found in fumes from anthracite or charcoal fires in the exhaust gas of petrol engines and in the gas ("after damp") formed in explosions in coal mines Carbon monoxide poisoning may thus occur as 1 An industrial accident in a coal mine explosion or as an industrial risk in the manufacture of ammonia the synthetic production of methane charcoal burning or carbide manufacture 2 A home accident from using an improperly ventilated bath geyser gas stove anthracite or charcoal stove or from the exhaust of a car whose engine is kept running in a closed garage 3 A means of suicide from a gas oven a gas fire or a car

**Pathology** Carbon monoxide combines with avidity with hæmoglobin in the blood forming carboxy hæmoglobin and displacing oxygen Anoxemia thus ensues Poisonous symptoms such as malaise and headache may be expected if the blood is 30% saturated with CO Unconsciousness ensues with 50 to 55% saturation and in fatal cases the blood is usually about 80% saturated Some patients die from nervous lesions, even although all CO has been removed from the blood Post mortem The face and lips are pink the blood is cherry red and minute hæmorrhages may be found in the brain lungs and other organs There is usually œdema of the lungs Bilateral degeneration of the globus pallidus (Kolisko s lesion) and cerebral œdema may be found

**Clinical Findings** *Acute Poisoning* The patient is usually discovered unconscious with stertorous breathing frothy exudation on the lips injection of the conjunctivæ dilated and fixed pupils a pink colour in the face and lips a rapid pulse and low blood pressure

*Chronic Poisoning* The symptoms develop insidiously the patient probably not knowing that he is breathing a poisonous gas, although the carbon monoxide has a faint odour of garlic He suffers from lassitude headache giddiness palpitations and nausea there is a failure of mental powers the patient cannot read clearly and then finds he is unable to move his limbs Sensibility to pain is also abolished There is usually no dyspnœa 10 ml of blood should be removed from a vein and placed in a small tube corked and sent to a laboratory for examination for the spectrum of carboxy hæmoglobin

**Differential Diagnosis** The circumstances in which the patient has worked or is found usually give a good clue to the diagnosis which is established by detection of carbon monoxide in the blood

**Course and Complications** Death may rapidly ensue but the course depends upon the degree of saturation of the blood with carbon monoxide Bronchopneumonia and myocardial degeneration with premature systoles may occur as complications Sequelæ include confusional psychoses and a Parkinsonian syndrome

**Prognosis** This is very grave in acute cases unless the patient receives adequate treatment before the carbon monoxide in the blood rises over 50%

**Treatment** *Prophylactic* Care should be taken that geysers and stoves have adequate flues and that motor cars are properly venti

m 180 by mouth or ½ to 1 fl oz by rectum Alternatively Medinal gr 15 may be given by mouth, followed by gr 5 every 4 hours Alcohol should be stopped at once Good results may follow the intravenous injection of 100 ml of 50% dextrose solution accompanied by the intramuscular injection of 20 units of soluble insulin Aneurine hydro chloride, mg 100 should be injected intramuscularly If there is evidence of congestive heart failure digitalis should be given The patient should be encouraged to take as much fluid as possible, especially milk

## Methyl Alcohol Poisoning

This is more likely to occur in countries where there is prohibition The chief symptoms are giddiness dyspnœa, nausea and vomiting Optic atrophy is prone to ensue It is a powerful poison and liable to cause death

**Treatment** The patient should be given 1 fl oz of castor oil and an intravenous injection of a pint of a 5% sodium bicarbonate solution to combat acidosis The effect and dosage can be checked by the determination of the alkali reserve of the blood

## Benzene Poisoning

**Etiology** Benzene poisoning is an industrial disease which may occur in distillers of coal tar manufacturers of benzene mixtures for motor cars in rubber manufacturers and in workers applying paint by the spray process

**Pathology** *The post mortem appearances in acute benzene poisoning* resemble those of asphyxia with hyperæmia of the various organs

**Clinical Findings** *Acute Poisoning* This results from inhalation of air containing a high concentration of benzene The individual becomes dizzy and rapidly collapses and death may occur in a few hours

*Subacute and Chronic Poisoning* The subject becomes weak and suffers from headache, a severe and progressive anæmia of an aplastic type develops in which there is a progressive diminution of both red and white cells Severe hæmorrhages may occur into the skin and from the nose mouth and intestinal and genito urinary tracts

**Differential Diagnosis** The nature of the patient s occupation, the typical anæmia and leucopenia establish the diagnosis

**Course and Complications** The course is progressive unless the source of the poison is removed

**Prognosis** This depends upon the concentration of benzene in the air inhaled

**Treatment** *Prophylactic* In dangerous occupations adequate ventilation should be enforced Clothes gloves and boots must be taken off on leaving work and cleaned at frequent intervals Baths should be taken daily Alcohol should be avoided Workers should be subjected to monthly blood counts

*Curative* In acute poisoning artificial respiration and the administration of oxygen are required A blood transfusion should be performed in all cases showing marked blood changes or hæmorrhages

to the degree of coma present If there is no improvement as judged by slight movements moaning or deeper respirations the intravenous injection is repeated at intervals of 15 minutes Slight twitching of the facial muscles indicates that a sufficiency of the picrotoxin has been given The picrotoxin can be subsequently injected intramuscularly every ½ to 1 hour when the optimum effect from the intravenous injections has been obtained Pentothal Sodium (1 G in 10 ml of distilled water) should always be at hand for intravenous injection the dose being 0 3 G should convulsions ensue from an overdose of picrotoxin Further points in the treatment are concerned with the bed being warmed and its foot tilted 6 to 12 inches on blocks an airway should be maintained and pharyngeal secretions removed by suction The danger of picrotoxin is that it may lead to convulsions while the patient is still in coma and this may require the intravenous injection of barbiturates to control them If the patient is dehydrated or if there is suppression of urine isotonic saline containing 5% dextrose should be given by intravenous drip or by continuous gastric drip One to two pints may be given intravenously in the 24 hours but a watch must be kept that œdema of the lungs does not occur An alternative to the picrotoxin treatment is the intravenous administration of amphetamine (Benzedrine) sulphate mg 10 of amphetamine sulphate in 1 ml of isotonic saline are injected intravenously without further dilution In severe cases the initial dose may be mg 40 with subsequent injections of mg 20 every half hour This causes an immediate rise of the blood pressure and an increase in the pulse rate In a moderately severe case mg 40 may be given over a period of 4 to 5 hours As an alternative to the analeptic treatment barbiturate antagonists may be given These are bemegride (Megimide) and amiphenazole (*Daptazole*) *used in combination Megimide is the more active of the* two rapidly restoring a patient from deep coma to a state of light anæsthesia which is comparatively safe thus abolishing the need for strict and prolonged medical and nursing attention Daptazole is a synergist to Megimide and also a good respiratory stimulant The drugs are given in normal saline 0 5% Megimide and 1 5% Daptazole A 5% intravenous dextrose drip transfusion is set up the two saline solutions of the drugs are put in separate 20 ml syringes Every 3 to 5 minutes 1 ml of the Daptazole solution is injected into the rubber tubing of the drip followed immediately by 10 ml of the Megimide solution After each injection the pulse and respirations are counted the blood pressure is taken and the reflexes are tested The injections are continued until the patient is brought to a safe state as shown by a return of muscle tone and reflexes including the pharyngeal and laryngeal For a deeply comatose patient the treatment takes about 2 hours A total dose of about 200 ml of the Megimide and 20 ml of the Daptazole solutions are required in most cases

It is very difficult to judge the value of these forms of treatment Nillson in Sweden in 1951 treated 176 cases without any analeptic drugs the mortality rate of 1 7% being much lower than the average mortality rate of cases treated with analeptics Locket in 1956 reported

lated In suspected atmospheres a *canary in a cage forms a good danger* signal, as it is very sensitive to carbon monoxide, and will fall off its perch if exposed to small concentrations of the gas

*Curative Acute Poisoning* The patient should be taken into the open air and artificial respiration given by the Schäfer or Eve method The patient is placed prone, with a thick rug or coat under the lower ribs and epigastrium The operator kneels across the patient's body, facing the head, and places his hands on the *lower ribs* *The operator by* bending forward and throwing his weight on his arms, slowly presses on the lower ribs and so expels the air from the lungs He then gradually relaxes his pressure by bringing his body upright again, and allows the lungs to re expand, without removing his hands from the patient s chest This process is repeated twelve to fifteen times a minute As soon as available, oxygen, or better, oxygen containing 7% carbon dioxide should be administered through a B L B mask The administra tion of $CO_2$ and oxygen combined will rid the blood of CO four times as quickly as will the inhalation of oxygen alone In the meanwhile the patient must be kept warm and stimulants applied, such as 1 5 to 5 ml of Coramine injected slowly intravenously After apparent recovery the patient should be kept very still for several days, as move ment may cause heart failure If there are signs of myocardial damage the patient must be kept in bed for several months

*Chronic Poisoning* Recovery usually occurs rapidly if the patient is placed in a pure atmosphere

## Poisoning by the Barbiturates

**Etiology** The barbiturates or diureides include barbitone or Veronal (soluble barbitone) Medinal, Luminal (phenobarbitone) Luminal Sodium Dial Phanodorm Allonal Veramon Amytal Evipan Soneryl Nembutal etc They are used as hypnotics but in overdoses or owing to personal idiosyncrasies may produce coma Deaths from barbiturates in England and Wales were 53 in 1938 247 in 1948 and 574 in 1954

**Clinical Findings** In coma due to barbiturate poisoning the pupils are usually dilated and react to light (in very severe cases they are contracted and reactionless) and the tendon reflexes are often *exaggerated* *In some cases there is excited delirium* The urine output is much reduced The patient may remain in coma for several days before death or recovery takes place The temperature is high apart from pulmonary complications, in severe cases of poisoning Broncho pneumonia usually develops as a terminal event

**Treatment** *Prophylactic* The Pharmacy and Poisons Act of 1933 which came into force in 1936 restricts the sale of these poisons and they can now only be obtained on prescription

*Curative* Gastric lavage is best avoided Artificial respiration should be applied and oxygen with 7% $CO_2$ administered if there are signs of respiratory failure and an air passage maintained Picrotoxin is the most powerful analeptic drug It is given at first intravenously using a 0 3% solution (1 ml = 3 mg ) mg 6 to 12 are injected according

should be washed out, as it is excreted into the stomach Two gallons of warm water should be used containing pot permang gr 60 Lethidrone (Nalorphine hydrobromide) is a morphine antagonist It is put up in ampoules of 1 ml containing mg 10 of the drug In acute morphine poisoning mg 10 to 40 are injected slowly intravenously or subcutaneously the dose being sufficient to restore a satisfactory respiration rate The patient remains drowsy To stimulate the respiratory centre oxygen or oxygen and 7% $CO_2$ should be given as for carbon monoxide poisoning together with artificial respiration

## *Morphinism*

*(including Heroinism and Opium Addiction)*

**Definition** Chronic poisoning from opium or its derivatives

**Etiology** Morphine is taken by injection heroin by injection in a linctus or as snuff opium by mouth or by smoking *Predisposing causes* 1 Race Opium indulgence is common in India, China Persia and Turkey 2 Age and sex A habit is more easily established in women and in young people 3 Disposition Morphine addicts often have a pyschopathic or neuropathic tendency, but only a small proportion are degenerates 4 Pain and worry The drug may have been taken to relieve sciatica chronic rheumatism asthma bronchitis gastric or duodenal ulcer for over work or because of phobias of cancer or insanity 5 Occupation Doctors nurses and pharmaceutical chemists have greater facilities for access to these drugs A morphine habit is not usually established unless the drug is taken regularly for several weeks or months

**Pathology** There are no characteristic post mortem appearances and death is usually due to some intercurrent disease Often in cases of acute poisoning no morphine can be recovered from the body but in some instances it is present in the liver, kidneys etc

**Clinical Findings** The patient may give a history that he began to take the drug to relieve pain insomnia or worry, and that he has been unable to discontinue it He may be able to do good work and to play games on a fixed daily dose such as gr 6 to 8 of morphine He gains no pleasurable sensations from the drug and wishes to give it up but is unable to do so In some cases there is no indication that the individual is taking drugs in others tremors or other symptoms suggestive of drug addiction may be noticed In more advanced cases the patient s friends can give more information They will testify to a change in character and habits The addict tends to be secretive loses concentration and application is irritable and depressed except when under the influence of the drug He becomes careless in his habits as regards cleanliness and dress In some cases as much as gr 40 of morphine are taken daily

*On Examination* The patient may deny taking drugs or make a frank confession He may complain of alternating constipation and diarrhœa The nutrition is usually poor the complexion sallow the hands moist and the nails show trophic changes The pupils are usually

no deaths in 186 successive cases of barbiturate poisoning given no analeptic or 'specific drugs In 1 000 cases with no analeptic or specific drugs" the death rate was 1 8%, much lower than the mortality rate of those treated with these special drugs

## Aspirin Poisoning

**Etiology** Poisoning is usually the result of an overdose taken with suicidal intentions

**Clinical Findings** There is a latent period before the signs of poisoning appear which varies with the amount taken In one case gr 750 were taken the latent period being 14 hours and in another in which gr 1 250 were swallowed toxic signs appeared after 9 hours

*On Examination* The patient is usually sweating freely and there may be exophthalmos The pulse and respiration rates are raised and the temperature is subnormal The patient may vomit small quantities of blood stained fluid The pupils are small and may be fixed, and the deep reflexes are normal or depressed There may be mental confusion and the patient may be alternatively violent or drowsy and complain of irritation of the scalp In more severe cases the patient sinks into coma and there is extreme air hunger The urine gives a strongly positive purple ferric chloride reaction and contains salicylates There may be a trace of albumin The alkali reserve of the blood is reduced and the blood urea raised

**Prognosis** The prognosis depends upon the amount taken the patient s susceptibility and the treatment given Death has occurred from as little as gr 5 whereas recovery has followed the taking of gr 1 500 The average lethal dose has been estimated at between gr 450 and 600

**Treatment** If the patient is so restless that intravenous treatment is difficult morphin sulph gr 1/8 to gr ¼ should be injected sub cutaneously The stomach should be washed out with 2 gallons of warm water containing 2 oz of sod bicarbonate If the patient can swallow sod bicarb gr 30 should be given by mouth every 2 hours An intra venous injection should be given of 1/5 N saline containing 5% dextrose and 20 G sod bicarb in1 500 ml As soon as the alkali reserve rises to a normal figure the administration of sodium bicarbonate by mouth and intravenously should be discontinued If there is marked circulatory collapse 500 ml plasma should be injected intravenously and 4 ml Eucortone injected intramuscularly

## Acute Morphine Poisoning

**Etiology** Morphine poisoning may result from an accident an overdose being prescribed or dispensed or owing to idiosyncrasy on the part of the patient It may also be taken in an overdose with suicidal intent

**Clinical Findings** The patient is usually found comatose with small inactive pupils depressed reflexes a cold and clammy skin feeble pulse and weak respirations

**Treatment** Even if the morphine has been injected the stomach

**Clinical Findings** The patient may notice a bitter taste on swallowing the fluid containing strychnine Symptoms of poisoning rapidly ensue, muscular twitching passing into violent clonic and tonic convulsions *These may result in rupture of muscles or in opisthotonus*, emprosthotonus or pleurosthotonus The spasms pass off in a minute or so and complete flaccidity ensues between successive fits The pulse is feeble and frequent, and respiration may be interfered with causing asphyxia The mind remains perfectly clear and the dilated pupils and staring eyes express the torture of the individual Death is not usually delayed in fatal cases for more than an hour or so, and may occur in a few minutes

**Treatment** Powerful sedatives should immediately be administered such as a hypodermic injection of morphin sulph gr ¼, and the injection of thiopentone sodium or anæsthetisation with chloroform Thiopentone sodium should be given intravenously 2 to 3 ml of a 5% solution When the patient is under the influence of thiopentone or chloroform the stomach should be washed out with 2 gallons of warm water If there are signs of respiratory failure artificial respiration should be given together with inhalations of oxygen and 7% $CO_2$ When the effect of the thiopentone wears off a second dose can be given

### Acute Cocaine Poisoning

**Etiology** Poisoning may occur accidentally as the result of an injection of a 10% solution of cocaine prepared for the anæsthetisation of a mucous membrane or from swallowing cocaine from a naso pharyngeal plug Suicidal poisoning with death may result from an adult swallowing gr 34 of cocaine

**Clinical Findings** After taking a poisonous dose by mouth the patient notices dryness and burning in the mouth buzzing in the ears palpitations of the heart hammering in the head and general trembling There is headache the vision becomes impaired and cramps occur in the muscles The patient dies in about an hour

*On Examination* The pupils are widely dilated and the patient is pale

**Treatment** The stomach should be washed out with 2 gallons of warm water containing pot permang gr 60 Coramine 5 ml should be injected intravenously for respiratory collapse

### Chronic Cocaine Poisoning

**Etiology** The cocaine is usually taken as snuff injected hypodermically or chewed in the form of coca leaves The drug is taken for its stimulating effect Morphine and alcohol addicts may ultimately resort to it

**Pathology** Post mortem the liver kidneys spleen and lungs are congested but there is no pathognomonic sign of cocainism

**Clinical Findings** The victim derives a temporary sense of exhilaration followed by depression As the habit is established the craving for the drug becomes irresistible The addict is gradually

small, they may be unequal The tongue is furred and the breath is offensive The pulse and respirations are slow The deep reflexes are depressed and patchy areas of hyperæsthesia may be present, especially on the soles of the feet The sexual functions are depressed the urine may contain a trace of albumin The skin usually shows the marks of hypodermic injections and of old abscesses

**Differential Diagnosis** Whether or not a suspected person is a drug addict can readily be decided by isolating him so that he is unable to obtain a supply In an addict symptoms of deprivation appear in a few hours There is running from the eyes, yawning sneezing and restlessness In more severe cases the addict then becomes weak and trembling and suffers great agony He may have abdominal pain vomiting, diarrhœa and collapse become maniacal or comatose and finally die

**Course and Complications** The course is usually progressive as the addict is unable to break the habit, and gradually requires larger doses He will resort to any subterfuge to obtain it Complications include intercurrent diseases, such as septicæmia or pneumonia and infective hepatitis if several addicts use the same needle

**Prognosis** The addict cannot cure himself The prognosis depends upon the duration of the habit, the adequacy of the treatment and the underlying cause If the latter can be removed the chance of a permanent cure is greatly enhanced

**Treatment** *Prophylactic* The Dangerous Drug Acts of 1920–23 have made it more difficult for these habit forming drugs to be obtained and they should not be prescribed unless the patient will only need them for a short time or to relieve suffering in cases of incurable disease

*Curative* Patients should be treated in special institutions by physicians skilled in the appropriate methods Sudden withdrawal of the drug is still the best method of treating a young person who has only been an addict for a short time The acute withdrawal symptoms usually subside after 4 to 5 days During this time sedatives should be administered such as phenobarbitone gr ½ to 1, 2 or 3 times a day Hot baths and massage are also helpful adjuncts In cases of long standing addiction gradual withdrawal is usually necessary The morphine is reduced in stages injections being given at regular intervals the patient not knowing what dose he is receiving or when injections of saline are finally substituted for the drug Laughton Scott recommends that equal parts of tinc belladonnæ and liq ext hyoscyami be used at first 3 to 5 drops in water every hour the patient is awake The dose is increased to 15 to 20 drops hourly provided the pupils do not dilate Sleep is secured by increasing doses of phenobarbitone up to gr 6 at night Psychological methods form an essential part of the treatment

## Strychnine Poisoning

**Etiology** Strychnine poisoning is rare It may occur accidentally or be due to attempted murder

bottle or by inhalation Carbolic acid is frequently swallowed in attempted suicide

**Pathology** There is local sloughing and necrosis, especially at the lower end of the œsophagus and in the stomach

**Clinical Findings** The patient notices an immediate burning sensation on swallowing followed by anæsthesia Unconsciousness rapidly follows a large dose

*On Examination* A white mark may be seen around the mouth due to the caustic effect of the poison and the breath has a characteristic odour If the patient is unconscious the breathing is stertorous and bubbling with froth on the lips which are congested and swollen The face is cold and sweating The pulse is frequent and it may be weak and irregular Vomiting is unusual Rales may be heard scattered over the lungs The urine may be dark (see carboluria p 676) and contain albumin

**Course and Complications** In mild cases the patient recovers consciousness but appears dazed and the voice may be hoarse owing to *laryngitis* Complications include tracheitis, bronchitis broncho pneumonia lung abscess suppression of urine myocardial weakness portal and peripheral venous thrombosis and gastritis Œsophageal and gastric perforation are rare In severe cases death occurs in a few hours from respiratory and circulatory failure

**Treatment.** The stomach should be washed out with great care using a soft tube with 2 gallons of warm water containing 2 oz of *sodium or magnesium sulphate* There should then be left in the stomach 2 oz of this solution together with 10 oz of milk and the white of 1 egg The bed should be warmed with an electric cradle the foot of the bed raised 18 inches on blocks and the respiratory passage maintained Coramine 5 ml should be injected intravenously and repeated frequently if there are signs of respiratory failure Oxygen should be administered as required through a B L B or plastic mask

## FOOD POISONING

**Introductory** Ptomaine poisoning was the name formerly applied to poisoning by tainted food especially tinned meat and fish It was thought to be due to protein decomposition products produced by the action of micro organisms It is now believed that the majority of cases are due to infection with the micro-organisms themselves or their toxins and rarely if ever to ptomaines

### Meat Poisoning

**Etiology** Meat poisoning is usually due to infection with organisms of the Salmonella group these include the Salmonella enteritidis Bact choleræ suis Bact aertrycke S typhi murium and Bact para typhosum B Less frequently staphylococci are the infecting organisms in canned meat The meat may be infected by the hands of "carriers" of the organism or contaminated by the excreta of animals such as rats and mice The meat is not usually altered in appearance or in smell and toxins are not produced in the meat before ingestion This is however

unfitted for mental or physical work, loses his appetite suffers from insomnia and his muscles are weak and tremulous Hallucinations develop, and irritation of the skin giving rise to the sensation of creeping insects (cocaine bugs) This is felt chiefly on the palms There may also be delusions of persecution Sexual excitement is increased but sexual power is diminished

*On Examination* The pupils are usually dilated and the pulse frequent The nostrils may be inflamed, and acne occur on the face near the nose

**Differential Diagnosis** The general appearance of the patient suggests that he is taking drugs, the nature of which can only be found by close observation

**Course and Complications** The habit is usually progressive the patient may become insane

**Prognosis** The outlook is unfavourable There is often recurrence after treatment and death may be due to an overdosage or suicide by some other means

**Treatment.** *Prophylactic* The Dangerous Drug Acts of 1920–23 restrict the sale of cocaine

*Curative* The patient should be sent to an institution for drug addicts where the treatment resembles that given for morphinism

### Acute Atropine, Belladonna and Hyoscine Poisoning

**Etiology** Poisoning usually occurs accidentally in adults as from swallowing a chest liniment in mistake for a cough mixture in the dark A belladonna plaster may cause poisoning in some individuals Eye lotions containing atropine may cause acute poisoning especially in children Children may also eat the berries of the deadly nightshade

**Clinical Findings** The patient complains of burning and dryness of the mouth dysphagia nausea and diplopia

*On Examination* The patient may be found unconscious or in a state of excited delirium The face is flushed and an erythematous rash may be present The pupils are widely dilated and do not react to light The pulse is frequent

**Treatment** If the poison has been taken by mouth the stomach should be thoroughly washed out with 2 gallons of warm water or an emetic given and repeated in 2 hours if necessary A hypodermic injection of pilocarpine nitrate gr ½ should be given If there is respiratory failure artificial respiration and the inhalation of oxygen or of oxygen and $CO_2$ should be given as for CO poisoning Further stimulants should be administered *such as the slow intravenous injection* of 5 ml of Coramine and the rectal injection of 10 fl oz of strong hot coffee

### Phenol and Coal Tar Disinfectants Poisoning

**Etiology** These substances are available as a soapy emulsion *e g* Lysol which contains 50% cresols or as a plain solution *e g* acid carbol liq Poisoning may occur accidentally from dressings applied to wounds or from douches by swallowing fluid from the wrong

normal saline solution A dose of fl oz ½ of castor oil should then be given If the diarrhœa persists after the bowels have been well opened an astringent mixture should be given such as Bism carb gr 10 cret gr 15 tinc catechu m 20, aq menth pip dest ad fl oz ½ Fl oz ½ t d s a c Achromycin or aureomycin are of value in certain salmonella infections 50 mg /kg body weight in divided doses every 24 hours for 4 or 5 days

*If there is much abdominal pain* hot flannels or turpentine stupes should be applied and tinct opii m 10 can be added to the astringent mixture Collapse is treated by stimulants such as hot coffee (fl oz 10) per rectum Acidosis and dehydration are best combated by rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose every 4 to 6 hours The patient should only take small quantities of boiled water or albumin water during the acute stages later the diet is increased by adding dextrose (oz 4 in a pint of water) and citrated milk (gr 2 to oz 1) diluted to half strength with water rusks arrowroot cornflour jellies custard, thin bread and butter eggs and fish

## Botulism

**Definition** An acute variety of food poisoning due to a specific bacillus

**Etiology** Botulism is caused by the Clostridium botulinum (B botulinus) of which there are five types Type A produces the most virulent toxin The bacillus is a spore forming anaerobic organism occurring in the intestines of animals and in soil It produces an *exotoxin acting on the nervous system* This toxin *is present in the infected food* Any animal or vegetable food may be contaminated but such articles as sausages potted meat ham fish and canned meats and vegetables are most commonly affected The Loch Maree outbreak in 1922 was due to sandwiches containing wild duck paste The eight people who ate the sandwiches died

**Pathology** Post mortem hæmorrhages may be seen in the brain and spinal cord The heart muscle is soft and the lungs may show bronchopneumonia The toxin appears to paralyse motor nerve endings

**Incubation Period.** This is only a matter of hours as the toxin is present in the food

**Clinical Findings** The patient is taken ill after *eating some con taminated substance* and usually several people are affected who have partaken of the same food He feels ill with headache and usually complains of eye symptoms such as diplopia or blurring of the vision The mouth is dry and there is dysphagia and often nasal regurgitation of liquids The bowels are constipated *the legs and arms are weak*, but there is usually no pain and the mind is quite clear The voice may become very weak

*On Examination* The pupils are dilated and do not react Squint or nystagmus may be present and there is weakness of some of the external ocular muscles supplied by the third cranial nerve There

not so in the case of botulinus infection Mutton rarely causes meat poisoning, veal, pork and beef being the chief offenders Staphylococci may grow in salted meats and produce enterotoxins Improper preservation of canned meat insufficient cooking and delay in consumption of meat are predisposing causes of poisoning Children and debilitated adults are more prone to infection and outbreaks usually occur in hot weather Other diseases which may be conveyed by meat include infection with worms tuberculosis and rarely with anthrax Poisoning has occurred from eating duck's eggs contaminated with S typhi murium and since the use of dried eggs outbreaks of poisoning have occurred from infection with various Salmonella organisms, such as the S typhi murium, Newport Montevideo etc To minimise risk of infection from dried eggs the fluid mixture must be used immediately to prevent the organisms multiplying

**Pathology** Post mortem hyperæmia may be seen in the mucous membrane of the small intestine and to a lesser degree in the stomach

**Incubation Period** This is usually a matter of a few hours but it may be prolonged to 2 or 3 days

**Clinical Findings** The onset is sudden with shivering nausea vomiting and abdominal pain If the offending food passes into the intestine, diarrhœa follows The motions are at first loose and evil smelling and later they become watery and may contain mucus and blood

*On Examination* The tongue is furred the temperature often raised to about 101° F and an erythematous rash may appear In severe cases the patient becomes collapsed and cramps occur in the legs In some instances the causative organism can be recovered from the stools during the first few days, and a specific agglutination reaction is obtained with the patient s serum in the second week of the illness The urine may show acetone bodies if there is prolonged vomiting The vomit should be kept and examined for arsenic A specimen of the food should be retained for bacteriological examination

**Differential Diagnosis** The nature of the illness is usually suggested by the fact that more than one person who has partaken of the same food is affected Acute abdominal conditions, such as appendicitis should be excluded

**Course and Complications** The illness passes off in a day or so in slight infections in more severe cases the symptoms increase in intensity for several days A tendency to gastro enteritis may persist subsequently

**Prognosis** The vast majority of cases recover completely

**Treatment** *Prophylactic* Due care must be taken in canning food as regards the purity of the contents and the temperature to which they are exposed during the process The cans must remain hermetically sealed Blown " tins should be rejected, the contents of a tin should all be eaten the day it is opened

*Curative* The patient should be in bed and kept warm In the early stages the stomach should be emptied by an emetic of mustard and warm water (oz ½ in fl oz 10) or the stomach washed out with warm

*Malta Fever and Abortus Fever* This is conveyed by goat s and cow s milk

*Foot and Mouth Disease* Men may be affected by milk of cows suffering from the disease

*Milk Sickness* This is a disease which may affect man owing to consumption of the milk of cows suffering from the trembles

*Q Fever* This is described on p 589

The characteristics of a milk epidemic The onset is usually fairly definite and limited to a group of houses or a district supplied from one source The occupants of houses in which the milk is boiled are usually spared When scarlet fever or diphtheria is due to milk the symptoms are usually slight.

Treatment. *Prophylactic* The purity of the milk supply should be guaranteed by Public Health measures This involves inspection of cows and cowsheds cleanliness in milking pasteurisation rapid distribution and the use of sealed bottles In very hot weather milk *should be boiled directly it is received in the house and all milk* given to infants unless adequately pasteurised should be brought to the boil to lessen the risk of infection with tuberculosis

*Curative* This is considered in the sections dealing with the various diseases

## Staphylococcal Poisoning

In addition to infecting salted meats staphylococci may produce enterotoxins in milk cheese custard pastries ice cream liver sausage potato salad etc

## Fish Poisoning

Fish may be contaminated with the salmonella group of organisms Oysters are liable to infection also with the enterica group Mussels may cause poisonous symptoms owing to a toxin called mytilotoxin Diphyllobothrium latum infestation may be caused by caviare and by fish such as the pike carp etc

## Potato Poisoning

Sprouting potatoes may cause toxic symptoms This may be due to a poison called solanin produced by the action of micro organisms in the potato There is headache abdominal pain and gastro-enteritis

## Mushroom Poisoning

This is due to eating fungi which are mistaken for edible mushrooms Death may occur in a day or so *after severe gastro enteritis and* hæmoglobinuric nephrosis The Amanita phalloides (Death Cap) is responsible for more than 90% of deaths due to mushroom poisoning The essential poison it contains is Amanita toxin Anti phalline serum should be given if available in doses of 40 ml intramuscularly In addition mag sulph ½ oz should be given by mouth every hour until the bowels are opened after washing out the stomach with 2 gallons of water or normal saline For collapse symptoms an intravenous drip

may be bilateral ptosis and paralysis of the palate The tongue is furred and the temperature is usually subnormal The pulse is slow at the onset but becomes more frequent later There is flaccid paresis of the extremities and the deep reflexes are diminished No sensory changes are present The blood and cerebro-spinal fluid are normal, and the causative organism is not usually found in the stools

**Differential Diagnosis** The symptoms of botulism are somewhat analogous to those of belladonna poisoning If the infected food is given to chickens they develop paralyses Other forms of food poisoning, such as those due to infection with bacteria of the salmonella group must be excluded In the latter the chief symptoms are gastro intestinal There are no muscular pareses no visual disturbance and the temperature is usually raised

**Course and Complications** The acute stage of the disease is usually short, lasting 3 or 4 days Convalescence, however, is slow and disturbances of vision may persist for several weeks

**Prognosis** The mortality rate is high usually over 70%, and the shorter the incubation period the more likely is the disease to be fatal

**Treatment** *Prophylactic* The bacilli and spores are killed by heat such as 120° C for 6 minutes Foods when being canned should therefore be heated to an adequate temperature

*Curative* The patient should be kept in bed and the stomach immediately washed out with warm normal saline A polyvalent botulinus antitoxin if obtainable, should be given in doses of 50 000 units intramuscularly after sensitivity tests If signs of respiratory failure occur, the patient should be placed in a respirator An immediate injection of morphin sulph gr ¼ should be given as this delays the action of the toxin

## Diseases Carried by Milk

Milk forms a good culture medium for micro organisms and impure milk may cause much sickness The diseases conveyed by milk include

*Tuberculosis* Infection comes from the cows more rarely from human sources

*Diarrhœa* This is usually due to organisms such as Gaertner s bacillus and streptococci

*Tonsillitis* Streptococcal infection in milk may cause an epidemic of acute tonsillitis

*Enterica Group Infections* Milk is usually contaminated by the hands of a carrier or by infected water used for washing the cans or diluting the milk

*Cholera and Dysentery* The organisms usually gain access to the milk through infected water

*Scarlet Fever* Milk is probably infected from contact with milkers who have suffered from a mild unrecognised attack of scarlet fever or who are carriers of the disease

*Diphtheria* Infection here is usually from carriers of the disease

# INDEX

injection of normal saline containing 5% dextrose should be given using 3 or 4 pints To control pain and restlessness atropin sulph gr 1/100 and morphin sulph gr ¼ should be injected subcutaneously

### Cheese Poisoning

Infection with the salmonella group may occur, in some instances a toxin, tyro toxicon, may develop in cheese

### Rye Poisoning

The fungus claviceps purpurea may occur on rye or grains, causing ergotism Gangrene of the extremities or nervous lesions may develop

### Lathyrism

Vetch seeds, if used as a substitute for wheat, may cause symptoms of poisoning There is spastic paralysis of the legs with lumbar pain

### Food Idiosyncrasies

Certain individuals are sensitive to special articles of food Thus allergic symptoms may result from eating fish, eggs, milk etc An urticarial rash, nausea vomiting dyspnœa and collapse are the most prominent symptoms